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EAR - NOSE - THROAT

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THE
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VOL. XXXI ST. LOUIS, NOVEMBER, 1922 No. 11

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

FURTHER STUDIES IN THE FUNCTIONAL EXAMINATION OF THE ACUITY OF HEARING AND ITS RELATION TO THE PERCEPTION OF SOUNDS OF DIFFERENT PITCHES PRODUCED BY THE NEW ELECTRIC ACUMETER.*

DR. JOHN GUTTMAN, New York.

In the functional examination of our sense organs we are striving to ascertain principally two facts: (1) The amount of the specific function of the sense organ; in hearing, the amount of hearing power which the patient has at his disposal, and (2) in an affection of this sense organ, we endeavor by the functional examination to ascertain the seat of the affection, and as far as possible its treatment.

In most cases of diminished hearing, the patient is concerned primarily with his hearing power for the spoken word. But inasmuch as the measurement of the acuity of hearing for conversational speech is encountered with great difficulties, we have to resort to different substitutional methods, as whisper, watch, tuning fork, Galton whistle, monochord, etc. Before a gathering of this kind it is unnecessary for me to point out in detail all the inaccuracies and shortcomings of each and every one of the above-mentioned methods. Neither the initial intensity of the sound, nor its graduation is employed with any degree of constancy and exactness. None

*Read before the Section of Otology, New York Academy of Medicine.

of these methods alone indicates a dependable relation to the hearing power for speech. In order to arrive at a positive judgment of the hearing power for speech, we have to take into consideration the results of the combined employment of all these methods.

I therefore fully endorse the statement made by Dr. Hays in an article which appeared in the Annals of Otology, Rhinology and Laryngology in December, 1921, in which he says: "Tests of hearing acuity by means of tuning fork as well as by the watch, whisper and spoken voice are a part of our usual procedure. But the more I have recourse to such examination the less value I place upon it."

It is true that a normal ear possesses a superabundance of hearing power for ordinary social intercourse. A person with about one-half of normal hearing power is seldom considered hard of hearing or deaf for speech by his acquaintances, yet for diagnostic or scientific purposes it is very desirable that even small deficiencies be measured by the most exact and refined methods of examination possible. Only when our methods of examination will register even minute changes, can we hope that our diagnostic ability as well as our therapeutic measures will show some degree of advancement.

In a previous paper which I read last year before this section, I described an Electric Acumeter, by the use of which I could obtain more exact results than by the employment of present-day methods. The principal advantages claimed for the Electric Acumeter are: (1) that we can produce sounds of various pitches from about 50 to about 20,000 d.v. per second; (2) that we can very finely and minutely graduate the intensity of each pitch; (3) that we can reproduce with great exactness any pitch as well as intensity within the range of the instrument. None of the methods now commonly used, singly or in combination, can claim these advantages.

In addition to the above, the use of my instrument has the following further advantages:

(a) We can place the patient in a silent booth, thereby eliminating interfering outside noises, and thus render the examination more exact. This cannot easily be done in the commonly used methods, because there the graduation of the intensity of sound is effected by the variation of the distance of the source of sound from the ear, while in the acumeter the graduation of the intensity is accomplished by increasing or decreasing the amount of energy created by the electric current. The distance of the source of sound from the ear is very short and remains stationary.

The source of the sound being near to the ear and stationary, unilateral examination of the hearing power can be more easily ac-

complished than formerly, where in order to exclude the good ear from hearing, a Barany noise apparatus had to be inserted. A Barany noise apparatus, however, eliminates not only the perception of sound of the good ear, but interferes and to a great extent diminishes also the hearing power of the ear which is to be examined, thus causing inexactness of the examination.

(b) A switch which may cut out the electric current and thereby the sound, serves as a check upon the statements of the patient.

(c) With the use of a second telephone receiver the examiner may at all times listen in to the sound heard by the patient without interfering with the examination.

(d) The results obtained by the instrument can be graphically plotted on a chart, the abscissa designating the frequency of double vibration of sound per second, and the ordinate the intensity of same.

Where on account of a great diminution of hearing power the ordinary intensity of sound is not sufficient I produce a sound of greater intensity which is about four times greater than the normal, by eliminating some resistance in the apparatus.

As will be seen from the charts, a sound of the frequency of about 1000 d.v. per second is heard by the normal ear with the least degree of intensity, which must be increased as we proceed towards both ends of the tone scale.

The second great object of the functional examination as stated above, is the determination of the seat of lesion and its treatment. According to our present knowledge the disease can be located either peripherally in the sound conducting apparatus, or centrally in the sound perceiving apparatus, viz., in the labyrinth, cochlear nerve or brain centres. To come to a precise diagnosis, we again have to employ numerous methods of examination, because no single method will positively and without including several exceptions, point to a definite diagnosis.

The rule which is most constant and shows the least number of exceptions is, that we consider the cause for diminished hearing to be located peripherally in the sound conducting apparatus, when bone conduction is increased and the perception of low pitched sounds is diminished, whereas the diagnosis of a central affection is made, when bone conduction as well as the perception of high pitched notes is diminished.

The latter rule harbors numerous difficulties because diminished bone conduction compared to air conduction is encountered in a normal ear as well, particularly in older people, and the unilateral examination for diminished perception of high pitched sounds as

stated above, is almost impossible because the perception of sound of the second ear cannot be excluded satisfactorily.

The main reliance therefore for the diagnosis of the seat of the affection must be laid upon the precise measurement of the perception of the low as well as of the high sounds, which in the writer's opinion can best be accomplished by the electric acumeter.

In peripheral affections the demonstration of increased bone conduction will only corroborate the findings of the acumeter.

My limited experience proved to me that affection of the sound conducting apparatus will show not only a very decided diminution of hearing power for the low, but also for the high pitched sounds. Far advanced affections of the sound perceiving apparatus show a diminished perception of the high as well as of the low pitched notes. In the beginning of a central affection, however, the perception of the high pitched sounds will be more markedly affected, for nerves are more vulnerable and more easily injured.

To a similar conclusion came also Dr. Dean, who states, cochlear involvement is indicated when there is a diminution of the hearing power for the high notes, mare marked for the low.

As will be seen from the charts, there exists a fairly constant relation between the results obtained by the old method of examination and the results obtained by the electric acumeter. Diminished perception for the loud and whispered voice will usually correspond to a diminished perception of a sound with the frequency of about 1000 d.v. per second. Noises in the ear and diminished perception for the watch will usually coincide with diminished hearing for high notes. Rinne negative will coincide with diminished perception of low notes, provided that the factor of old age does not interfere.

I did not consider Schwabach's test, because I believe it is less reliable than Rinne's. In Rinne's test the comparison is made between the air and bone conduction of the same individual, while in Schwabach's test the bone conduction of the patient is compared with the bone conduction of a second individual, whose age, mental or physical condition may be responsible for the differences.

To summarize my experience as visualized by the charts, the following conclusions can be drawn:

(1) Diminution of hearing power for the spoken word will correspond to a lowering of the curve representing the whole field of audition, especially the sound with the frequency of about 1000 d.v. per second.

(2) Affections of the sound conducting apparatus will show a diminished perception mainly of the low pitched sounds, but the

high pitched sounds will also be frequently affected, though to a less degree.

(3) Affections of the sound perceiving apparatus will show a diminished perception of high pitched sounds, and in advanced cases of the whole field of audition.

(4) There exists a correlation of the hearing power of the two ears. It is well known that, as Politzer says, "the perception of the cochlear nerve for weak intensities is very changeable." Repeated examinations therefore may not always give uniform results, still the relation of the hearing power of the two ears remains remarkably constant, possibly because the underlying cause for the change is identical.

(5) Permanent perforations of the drum membrane, as well as retracted drum membranes caused by organized exudates with connective tissue formations in the tympanic cavity as seen in progressive o.m.c.c., produce very great diminutions of hearing power.

(6) In a case of sequellae of o.m.p.c. with a dry tympanic cavity, the hearing will be more diminished than in a case of o.m.p.c. where the purulent process is still active with succulent granulations.

(7) Calcareous deposits on the drum membrane do not always materially diminish the hearing power.

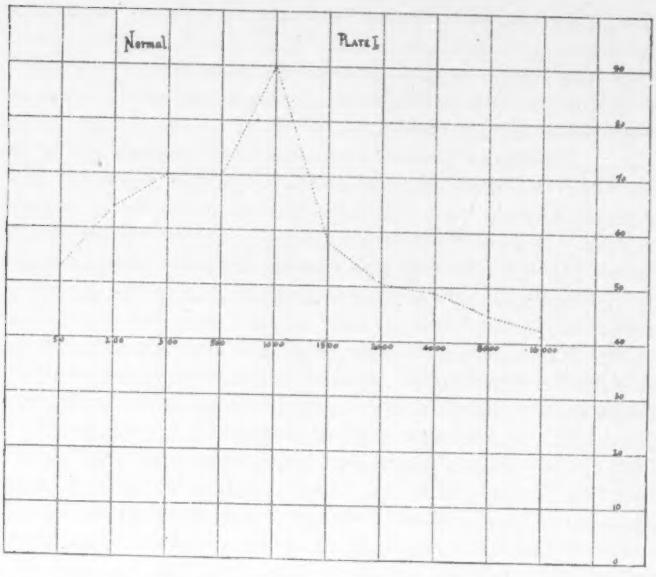
(8) Nerve deafness is sometimes combined with catarrhal deafness when Rinne will be found to be negative.

(9) Otosclerosis sometimes shows a very great diminution not only of the low but of the high notes also.

It is admitted that repeated examinations may give varying results owing to physical or mental changes on the part of the patient. It is believed, however, that the above mentioned method of examination gives promise of yielding more exact information than we have been able to obtain hitherto, and with more study and experience not only our diagnostic but also our therapeutic ability will be amplified in this special branch of otology.

Conventional signs used in plates: V, conversational voice; v, whispered voice; h, watch; W, Weber; R, Rinne; mt, membrana tympani; Au, both ears; AD, right ear; AS, left ear; Ac, ad concham; 10' $\times$ - more than 10 feet; M, male; F, female. Fraction following V and v designates feet. Fraction following h designates centimetres. The numerator of the fraction indicates the right ear. The denominator of the fraction indicates the left ear.

In the curve the solid line represents the hearing power of the right ear, the dashed line the hearing power of the left ear, and the dotted line the normal hearing power.


Normal
Plate I.

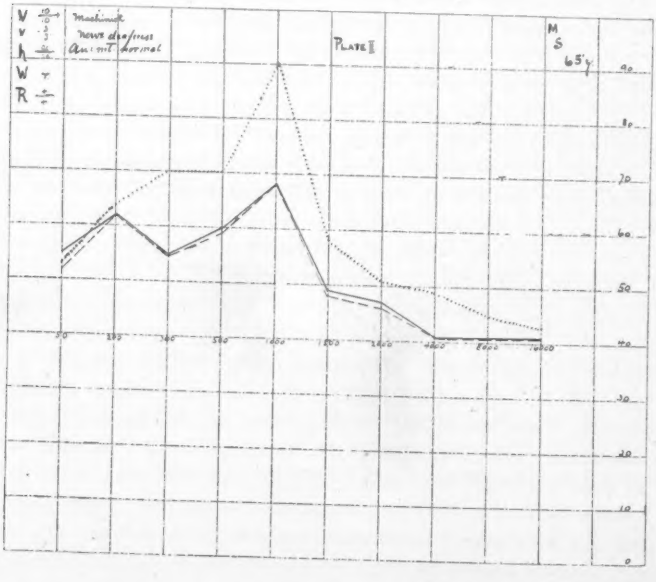


Plate II

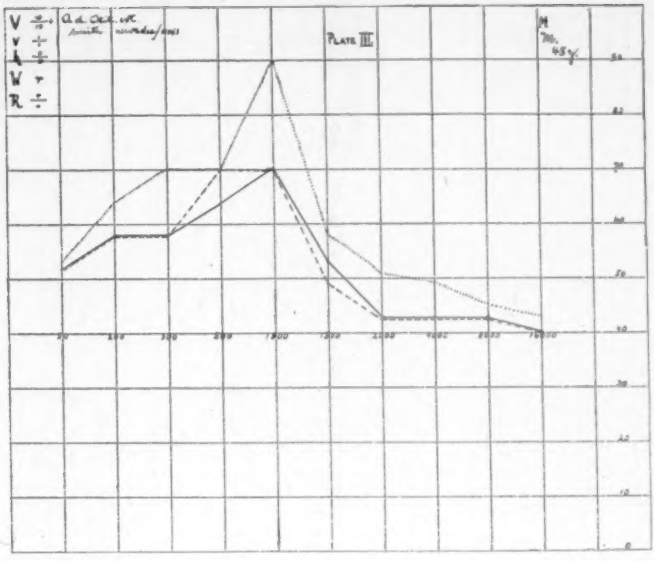


PLATE III.

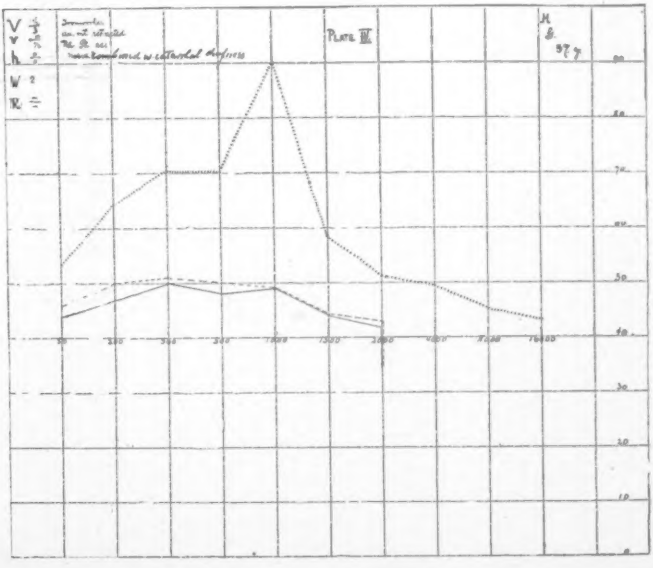
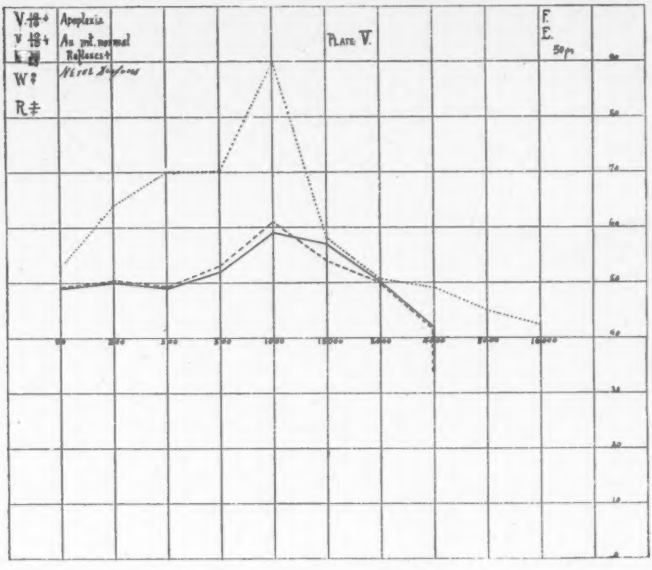
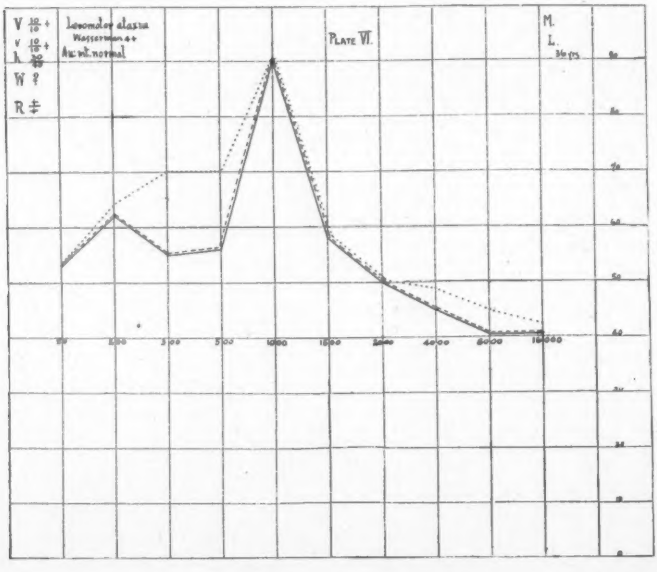
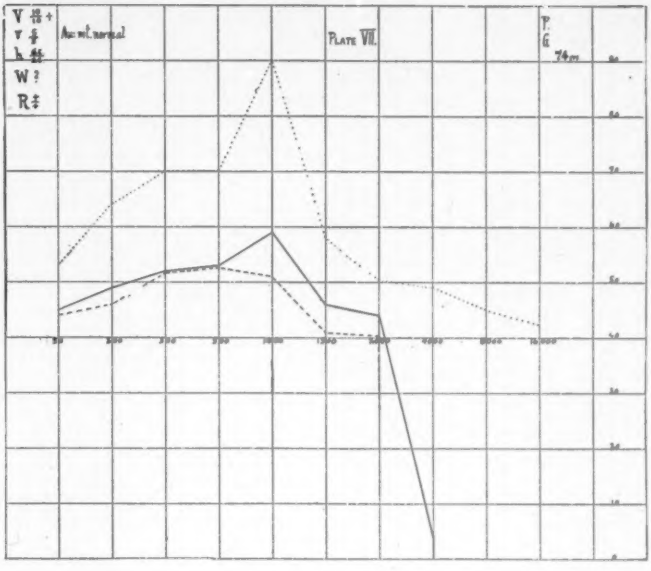
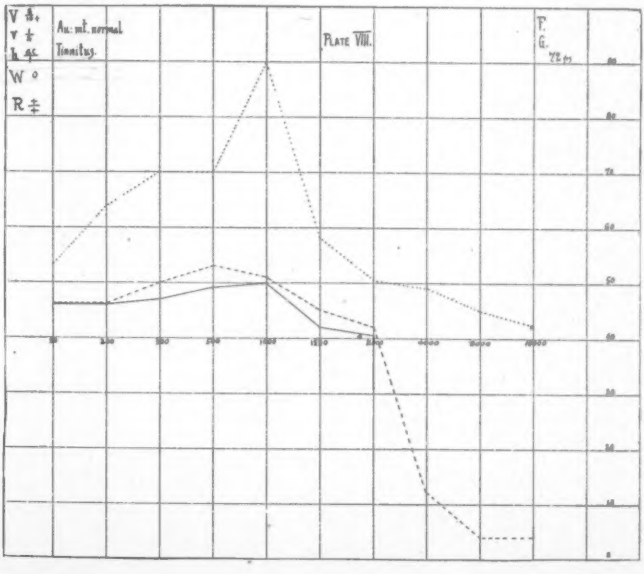


PLATE III.


Apoplexia
An. wk. normal
Reflexes +
Plate V.


Locomotor ataxia
Wassermann ++
An. wk. normal
Plate VI.


Au: ml. normal
Plate VII.
W ?
R ‡


Au: ml. normal
Tinnitus.
Plate VIII.
W o
R ‡

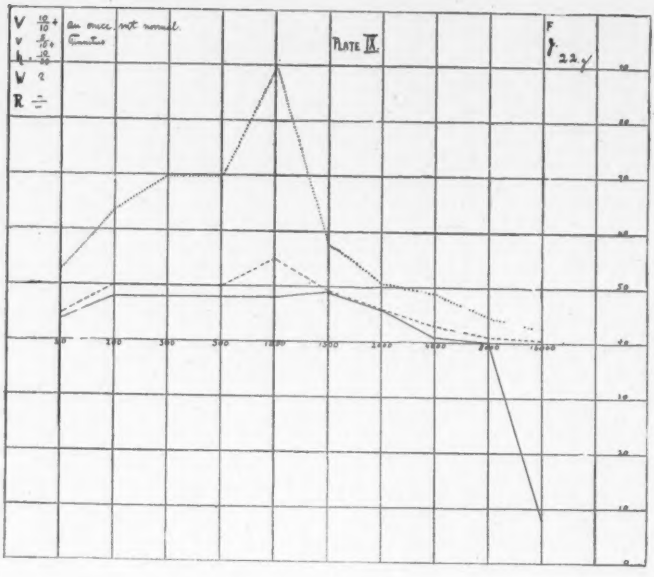


PLATE II.
F
22 y

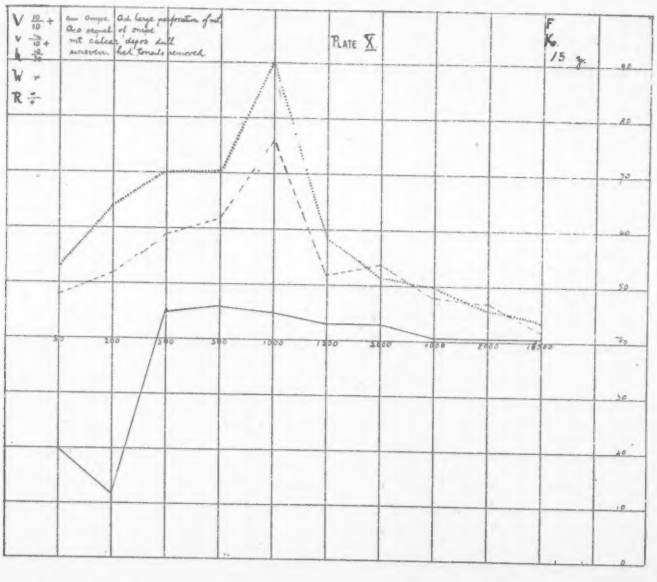


PLATE V
F
15 y

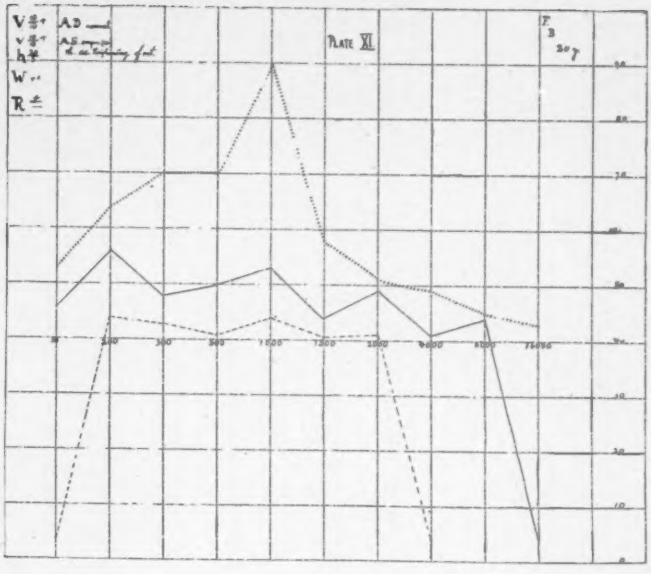


PLATE XI

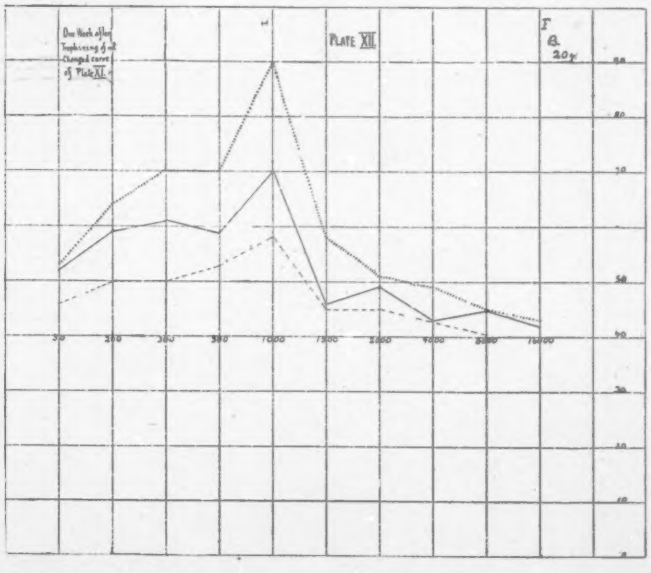


PLATE XII

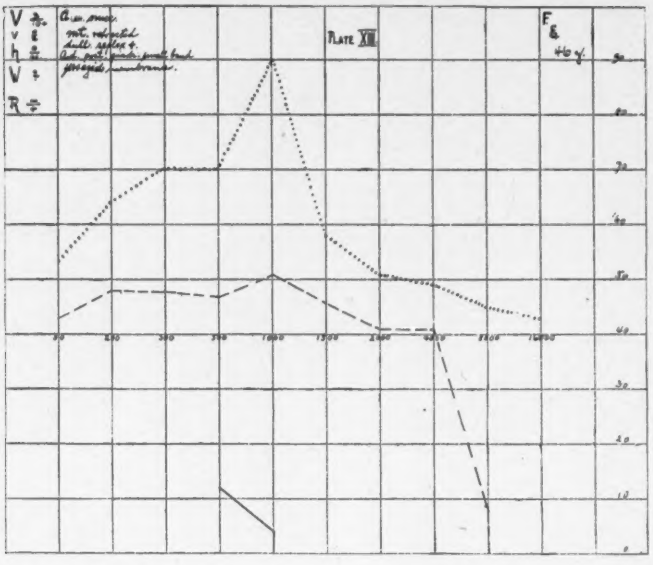


PLATE XII
F.
40 y.

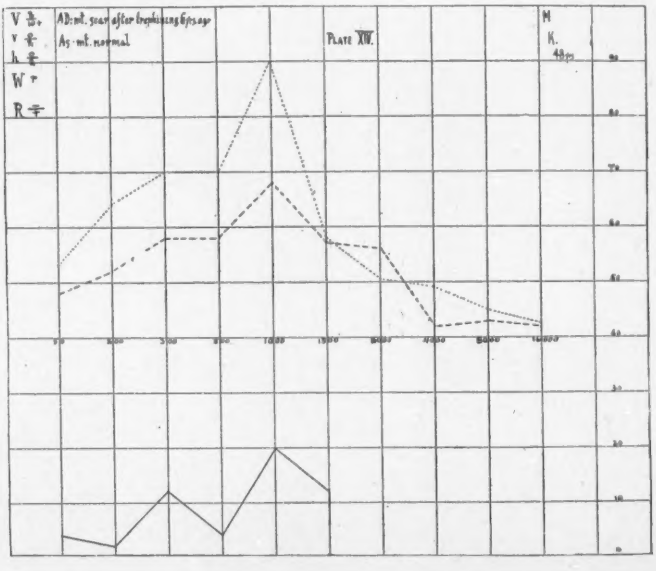


PLATE XIV.
M
K.
48 yrs

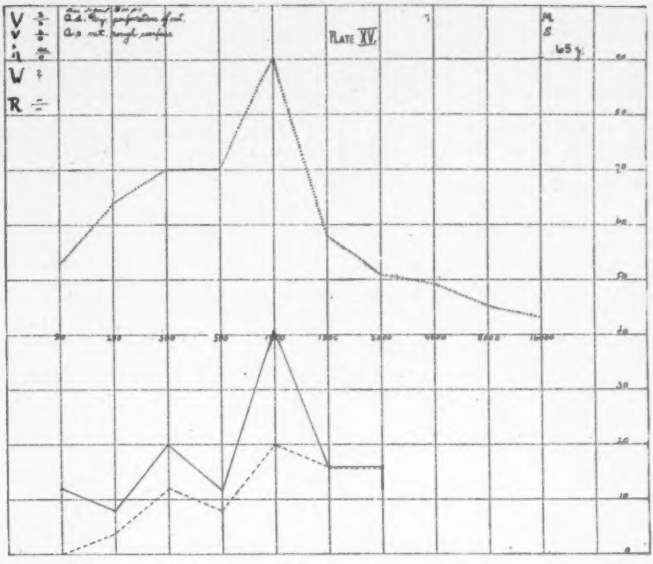


PLATE XV.

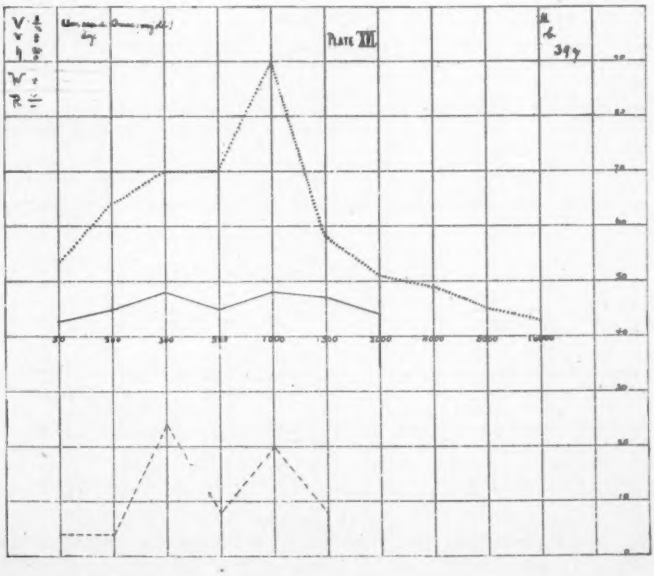
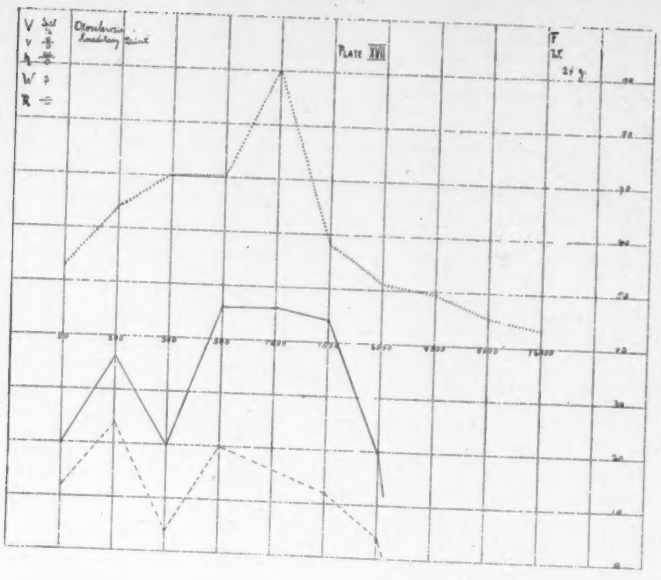
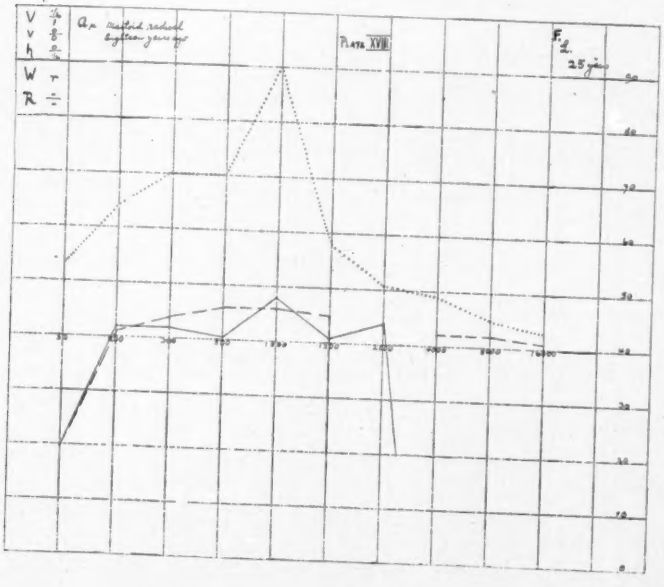


PLATE XVI.





EXPLANATION OF PLATES.

PLATE I.

Represents the normal curve obtained by averaging the results of examination of a number of persons with normal hearing.

The abscissa represents the number of double vibrations per second and the ordinate the acuity of hearing, which is in inverse ratio to the intensity. The minimum of hearing power is at 0. The peak of the curve is at about 1000 d.v. per second, showing that this pitch can be heard by the normal ear with the least amount of intensity.

PLATE II.

Diminished perception of pitch 1000 confirmed by:

$$\frac{3}{v\frac{}{3}}$$

Diminished perception of high notes confirmed by:

$$\frac{ac}{h\frac{}{ac}}$$

Normal perception of low notes confirmed by:

$$\frac{+}{R\frac{}{+}}$$

PLATE III.

Diminished field of audition confirmed by:

$$\frac{1}{v\frac{}{1}}$$

Diminished perception of high notes confirmed by:

$$\frac{o}{h\frac{}{o}}$$

Perception of low notes not much diminished confirmed by:

$$\frac{+}{R\frac{}{+}}$$

PLATE IV.

Curve of right ear below that of left ear as well as the reduction of the whole field of audition is confirmed by:

$$V\frac{\frac{1}{2}}{3} \quad \text{and} \quad v\frac{o}{\frac{1}{2}}$$

The lack of perception of upper notes above pitch 2000 confirmed by:

$$\frac{o}{h\frac{}{o}}$$

Diminished perception of low notes confirmed by:

$$R\frac{}{}$$

PLATE V.

Diminution of whole field of audition, especially the diminished perception of high notes, does not coincide fully with the examination by the ordinary methods.

Diminution of low notes not sufficient to overcome factor of old age, therefore

$$\frac{+}{R\frac{}{+}}$$

PLATE VI.

Slight bilateral diminution of upper notes may be pathognomonic for syphilis.

PLATE VII AND PLATE VIII.

Both plates show almost identical curves, due probably to the similarity of the ages of the patients.

The inferior position of the curve of right ear below that of the left ear in Plate VIII is confirmed by:

$$V\frac{8}{10}, \; v\frac{1}{2} \text{ and } h\frac{ac}{1}$$

Diminished perception of low notes cannot overcome factor of their ages, therefore

$$R\frac{+}{+}$$

PLATE IX.

Right curve below that of left curve confirmed by:

$$V\frac{5}{10} \text{ and } h\frac{10}{30}$$

Diminished perception of high notes and tinitus confirmed by:

$$h\frac{10}{30}$$

Diminished perception of low notes confirmed by:

$$R\frac{-}{-}$$

PLATE X.

Diminished field of audition, especially of right ear, confirmed by:

$$V\frac{½}{}$$

Diminished perception of high notes on right ear confirmed by:

$$h\frac{10}{}$$

Diminished perception of low notes on right ear confirmed by:

$$R\frac{-}{}$$

Calcareous deposit on the left drum membrane did not affect very materially the hearing power of that ear.

PLATE XI.

AD: slightly diminished field of audition, probably caused by acute rhinopharyngitis.

AS: Diminished perception of high notes confirmed by:

$$h\frac{}{3}$$

Diminished perception of low notes confirmed by:

$$R\frac{}{-}$$

PLATE XII.

Represents the condition of the same ears as in Plate XI one week later and after having established a better drainage by trephining the left drum membrane. The perception of low as well as of high notes improved.

PLATE XIII.

AD: Hardly any perception of sound present as V^2— probably heard by left ear.

AS: Diminished field of audition confirmed by:

$$v\frac{}{2}$$

Diminished perception of upper notes confirmed by:

h—
ac

Diminished perception of low notes not sufficient to overcome factor of age, therefore

R—
+

PLATE XIV.

AD: Practically no hearing in this ear.
Lack of perception of low notes confirmed by:

–
R—

AS: Diminished perception of low notes not sufficient to overcome factor of age, therefore

R—
+

PLATE XV.

AD: Diminished field of audition and diminished perception of high notes confirmed by:

2 2 ac
V— v— and h—

Great diminution of low notes cannot be overcome by age, therefore

–
R—
–

AS: Hardly any perception of sound present.

PLATE XVI.

AD: Diminished field of audition confirmed by:

8 O
V— v—

Diminished perception of high notes confirmed by:

ac
h—

Diminished perception of low notes confirmed by:

–
R—
–

AS: Very little perception of sound present.

PLATE XVII.

AD: Diminished field of audition confirmed by:

½ o
V— and v—

Diminished perception of high notes confirmed by:

ac
h—

Diminished perception of low notes confirmed by:

–
R—

AS: Very little perception of sound present.

PLATE XVIII.

AD: Practically totally deaf.
AS: Shows a tone gap between tone pitch 2000-4000. The slight perception of the sound above 4000 is confirmed by:

h—
½

Diminished perception of low notes confirmed by:

–
R—
–

616 Madison Avenue.

ACUTE MASTOIDITIS, COMPLICATED BY AN ACUTE CONCURRENT DISEASE.*

DR. C. F. ADAMS, Trenton, N. J.

Many acute diseases are complicated by the involvement of the middle ear and mastoid. Influenza, tonsillitis, typhoid fever, pneumonia, the exanthemata, tuberculosis, syphilis, erysipelas, etc. It is more unusual to have a mastoiditis begin at the same time as the other acute disease and run, apparently, an independent course.

Dr. L. M. Huddy, in the annals of Otol., Rhinol. & Laryngol., Vol. 25-1030, 1916, reports the case of a man, 36 years old, who had influenza 15 days before he developed an acute suppurative otitis media with mastoiditis; at the same time the urine contained 3.5 per cent sugar, temp. 104. Mastoidectomy was performed; for 72 hours the above condition continued; at the end of this period the temperature fell by lysis, sugar disappeared from the urine on the fourth day after the operation and the patient gradually returned to normal.

Dr. G. F. Keyser, in *Laryngology*, Vol. 25-28, 1915, reports two cases of mastoiditis, complicated with acute nephritis, one of which recovered from both diseases. The second succumbed from the nephritis.

We know that the kidneys are frequently affected in almost all diseases that have a high temperature, and especially in scarlet fever, measles and erysipelas.

Dr. J. A. White, in *Laryngology*, Vol. 14-689, 1904, cites a case of a boy, aged 7 years, upon whom he did a simple mastoidectomy, discharging him from the hospital in two weeks. Ten days later he had a sudden accession of fever and discharge of pus from the antrum. This continued for three weeks, when the urine became scanty and of a dark color, the mastoid became red and swollen. The next day the swelling back of the ear proved to be erysipelas. This condition continued with a high temperature for a week, then a small feruncle was found, just over the antrum, which was punctured, a small bead of pus obtained, after which healing and recovery was prompt.

I am reporting three cases of acute mastoiditis with acute concurrent diseases:

First: Otitis media, suppurative, unilateral. Concurrent diseases, pneumonia, meingitis.

M. P., age 24, service 4 months. Admitted to base hospital January 12, 1918. Present history: Reported to regimental hospital

*Read before the Philadelphia Laryngological Association, Dec. 6, 1921.

infirmary January 12, 1918; sent from there to base hospital. He reported that he had had for 3 or 4 days an acute coryza and some cough, but was not sick enough to report on sick leave until the morning of the 12th. In the night he had vomited twice and had a chill with fever, bowels had not moved since January 10. On admission he complained of headache, pain in the chest, legs, arms and back; cough, fever, anorexia, constipation; the naso-pharynx congested, thick mucus, no membrane.

January 13. Diagnosis: Lobar pneumonia, left lower lobe. Temp. 104. Given 100 c. c.'s mixed pneumoccoccio serum three days in succession. January 26. Chest clear. January 31. Pain in joints.

February 1. Rash over entire body, joints stiff and sore. February 1, 5 p. m. Temp. normal. February 6, A. M. Pain in left ear and down neck. Temp. 100.6. Restless during night. February 6, 3 p. m. Nose and throat negative; left tympanic membrane red and bulging. Diag.: Acute otitis media, suppurative, unilateral left. Treatment: Myringotomy. Free discharge of serum. Swab sent to laboratory. Laboratory diagnosis: Streptococcus hemolyticus. February 14, 9 a. m.: Temp. 103. Moderate drainage of serum from left ear, acutely delirious when disturbed; sleeps at other times. Major Palfrey reported some resistance to forward bending of neck and slight Kernig's with severe delirium, indicates lumbar puncture.

February 14, 2:40 p. m. Lumbar puncture by Major Palfrey; about 40 c. c. cloudy fluid under pressure withdrawn. Bacteriological findings: Pneumocci, type iv. Treatment: 20 c. c.'s anti-pneumococcic serum and 20 c. c.'s streptoccus serum introduced by gravity. Morphia and atropine given before puncture.

February 14, 4:45. Expired.

Diagnosis: Acute pneumoccocic meningitis.

Pathological findings. Sinuses clear. Left mastoid congested. Meniges of frontal lobes covered with exudate. No connection found with sinuses or mastoids. In this patient, within a month, there were found three acute affections, due to different organisms, viz: 1. Pneumonia-pneumoccocci, type 1. 2. Acute otitis media suppurative. Streptococci hemolyticus. 3. Meningitis, pneumococci, type 4.

The lung and ear affections probably were carried through the air passages, and the meningitis through the blood. The serum treatment was neither prophylactic or curative in this case.

Case 2. Acute mastoiditis. typhoid fever, concurrent disease. R. T., male, age 14, 1921. Family history: Father, mother, two brothers and sister living and in good health. Previous history:

When five years old had pneumonia, later influenza and measles. Has had tonsils and adenoids removed. Five months ago had small bone removed from nose because of difficulty in breathing. This operation was followed by an attack of chills and fever. Present trouble began 4 or 5 weeks before admission to the Mercer Hospital, Trenton. He complained of pain in the right ear, loss of appetite, fever and a dry cough.

September 4. Admitted to hospital. Temp. 105, dizziness, nausea and vomiting. The mastoid was sensitive to pressure, tympanic membrane; dull reflex and bulging.

Under chloroform anesthesia a free myringotomy was performed and a small amount of pus was obtained. He was returned to bed and was given a dose of calomel, followed by a dose of mag. sulp. Urine was 1030 sp. gravity with trace of albumen, many bacteria and amorphous urates. The discharge from the ear contained streptococci and staphlylococcaureus. No other organism.

September 6. As there was no relief from the pain in the ear and dizziness, under ether anesthesia the mastoid bone was opened. The process contained large cancellated cells throughout, filled with serum, blood and pus, not much necrosis. The antrum and additus were large. The bony process was thoroughly exenterated, lightly packed and a suture placed in the skin. A gauze drain was also inserted in the external meatus.

September 7. Temp. 101, no pain, dizziness or nausea. This temperature continued for a week, but on the 13th it rose to 103, dropping in the morning to 100.5, no pain or tenderness about the ear or neck, wound clean; packing and sutures were removed on the 5th day.

September 16. Ten days after the operation temp. rose to 104, no chill. Blood examination: No malaria parasites. W. B. C., 12,300; polys, 649 or 62.2 per cent; lymph, 149 or 14.2 per cent; L. mono, 169 or 16 per cent; trans., 49 or 4.7 per cent; megalocytes, 29 or 2.9 per cent. Urine negative. Wassermann negative. The temp. see-sawed up and down from 100 to 103 till September 26, or the 20th day after the operation. During this period the abdomen was flaccid, bowels inclined to constipation, but moved freely with a soapsuds enema and were not suggestive of anything out of the ordinary.

September 21. Fifteen days after the operation, his blood was examined for typhoid fever and gave a positive Weidel reaction. About this time he developed hallucinations, was afraid of being poisoned. This continued for a month after all other symptoms had disappeared. He finally made a good recovery.

It was found later that the adjoining neighbor's family had also contracted typhoid fever.

Case 3. Acute mastoiditis. Acute nephritis, concurrent disease. R. D. H., male, age 14, weight 78½. September 26, 1921. Family history: Mother died of pneumonia. Father living and in good health, was subject to attacks of earache, followed by a discharge, all his life until 4 or 5 years ago. Ears are now dry, but acuity of hearing reduced 50 per cent. Three brothers and sister living and in good health, all subject to attacks of earache. Tonsils and adenoids removed from all the children.

Personal history: Student. Well nourished. Right ear has discharged every summer after being in swimming.

September 4. Present trouble began. Was very dizzy, severe headache, temp. 105, discharge from right ear. Was sent to the Mercer Hospital, Trenton, September 5, by the family physician and prepared for operation the next day, September 6. Urine was a dark amber, heavy precipitate, sp. grav. 1020, reaction neutral, albumen positive, casts hyaline, large and small, fine granular blood casts. W. B. C., 30-100 to H. P. F.; R. B. C., 10-30 to H. P. F. Uric acid crystals, urea oxalate crystals. Bacteria + +.

Right mastoid process was opened, the cortex was hard, few cancellated cells, the antrum was exposed, all diseased bone was removed, wound was lightly packed with gauze, the upper 2/3 of the skin incision was closed with three sutures and a small piece of gauze inserted into the external aural meatus. At the time of the operation the temp. was 103, the next day the highest point was 100.6, on the third day it was normal and remained so until he was discharged, September 28, or 21 days after the mastoidectomy. At this time the urine was normal, there was a small perforation in the drum membrane, but no dizziness, and the headache subsided with the fall of the temperature.

In the first case, there is a fair illustration of the impossibility of assigning a special variety of bacteria owing to the numerous kinds found, to a special disease.

The difficulty in the second case, mastoiditis with typhoid fever as the concurrent disease, was in the fact that the fever was characteristic of neither mastoiditis or typhoid fever, but suggested lateral sinus thrombosis. There being no other symptoms of that trouble but the fever it was excluded, and the diagnosis was finally made from the blood reaction for typhoid.

In the case complicated by nephritis, it was difficult to account for the etiological factor because there are so many diseases which may cause nephritis. I am unable to say to which diseases I should assign the fever and dizziness.

AURAL LESIONS AS A CAUSE OF SEVERE SYSTEMIC INFECTIONS.*

DR. THOMAS J. HARRIS, New York City.

The medical history of the 20th century already contains many fair pages, but none fairer than the page which records the achievements of surgery in combating lesions of the ear which lead to general infections. All of these have been accomplished in an incredibly short time. Modern otology is scarcely more than fifty years old, and it is less than half that time since the profession began to realize the importance of the teachings of Zaufal (1885) and Ballance (1890) as to the necessity of surgical interference in these conditions.

Dench of New York was among the first in this country to direct attention to the subject in a paper[1] read in 1902 describing the operative treatment. In the discussion which followed, several distinguished aurists present admitted they had never done the operation. Notable advance has been made in the intervening twenty years. Clinical data of extensive proportions, and numerous monographs on all phases of the subject, have been written. In this development American aurists have had a conspicuous part.

In the face of so many authoritative treatises and such widespread knowledge, we would feel great temerity in presuming again to discuss the subject, if it were not for the increasingly prevalent opinion held by those possessing the widest experience that the classical description of sudden chill—high temperature followed by marked remission as typical of the disease is misleading and tends to serious error in the frequent overlooking of the so-called atypical cases. This was forcibly stated by Tobey[2] at a recent meeting of the American Otological Society, when he said: "I feel justified in making the statement that the text book so-called atypical case is very rare . . . . . many deaths have undoubtedly been caused by the fact that the text book diagnosis has been apparently so simple and the consulting otologists are repeatedly called in to see cases in extremis."

Properly to understand the clinical picture of sinus thrombosis there must be a clear recognition of the pathology involved in the various stages of the disease. At the outset it should be made plain that what we are considering is strictly not sinus thrombosis, but

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sinus thrombo phlebitis. This important fact has been pointed out by Kerrison in his "Diseases of the Ear" and Braun[3] in his paper before this section on the "Pathology of Sinus Thrombosis. We are dealing, in a word, with a condition of pyemia, the result of an infection which has entered the system through the great venous channels of the head. On the course the infection pursues on entering the system, depends the nature of the symptoms or the absence of symptoms. A prerequisite of sinus infection is an acute suppuration of exacerbation of a chronic suppuration of the ear, although cases have been reported where the external auditory canal, as well as the labyrinth, was the place of origin. As to whether the acute or chronic suppuration is the more frequent cause, authorities do not agree.

Before we proceed to discuss the more usual conditions, mention must be made of the much more rare, primary thrombosis of the jugular bulb. This was first described by McKernon[4] in 1905, who at that time reported a number of cases which he had encountered. The condition occurs chiefly in children, and is characterized by high temperature and profound prostration without apparent cause in the ear. It is generally held that the infection is conveyed directly to the bulb from the middle ear through some dehiscence in the floor. The infection, except in primary bulb thrombosis, proceeds from the ear in one of two ways, either by contiguity—confluent or coalescent mastoiditis,—(and here it apparently makes no difference whether the bone is of the diploic or pneumatic type) or through the blood vessels by extension of a phlebitis (hemorrhagic or vascular type), in which case the sinus wall is not affected. In the extension by contiguity, the necrosis extends to the bone directly over the sinus where there may be a collection of pus in the form of the so-called peri-sinus abscess.

Haymann[5] has shown experimentally that a septic tampon is the most certain way of causing a clot. The phlebitis, theoretically at least, is in all cases limited at first to the outer wall, which as the result of nature's attempt to resist invasion, is wont to be found covered with granulations. Whether the infection penetrates to the inner wall, depends chiefly on two factors; the virulence of the organism, and the resisting power of the patient. In epidemics of influenza or measles, for example, with the individual's vitality lowered by the disease, extension to the inner wall of the sinus is apt to occur. On the other hand, it is a common observation that the advance inward is often checked by prompt and complete removal of the diseased bone or abscess adjacent to the sinus. With the in-

volvement of the inner wall, the development of a thrombus is wont to take place.

Four conditions (according to Haymann) especially favor clotting:

1. Slowing of the blood stream.
2. Increase of agglutinability of blood platelets.
3. Increase of coagubility of blood plasma.
4. Change in the vessel wall.

The clot is at first parietal and it becomes only later, if ever, complete or obdurating. It is or can be at the outset, at least in part, aseptic. The ends of the clot may be sterile when the center is teeming with bacteria. From this point, the course of the infection varies. As the result of clinical observations, and particularly the experimental work of Haymann just referred to, it has been shown that one of five outcomes, so far as the clot is concerned, may occur.

1. Death may take place from sepsis before any change in the clot takes place.
2. The clot may break down in the center and discharge through a necrosis of the outer wall into the mastoid.
3. The clot may break down and discharge through the visceral wall, causing a cerebellar abscess.
4. The sinus may become obliterated.
5. Part of the clot may become absorbed, and the patency of the canal re-established.

Symptoms. The long time held division of typical and atypical cases must be given up. As Barnhill has said, "Every case of sinus thrombosis is a case by itself and may present difficulties of diagnosis well-nigh impossible to surmount." This is easily understood in the light of repeated case reports showing the exceedingly transient involvement of the middle ear and the *absence* of all *pain* in the mastoid—so-called *"painless mastoiditis."*

Lewis[6] and Fridenwald[7], among others, have reported cases where there was a history of slight discharge from the ear some weeks previous to the onset of the trouble, but at the time of the symptoms no discharge whatever was present. We saw such a case which we presented at the meeting of the American L. R. and O. Society, in New York in 1901. It was a man about twenty-three years of age, who had been admitted to the hospital April 3, with a history of pain and otorrhea for about one week and a swelling in the neck. There was no elevation of temperature at the time, and he complained only of frontal headache. A few days later an exploratory incision was thought to be necessary, and accordingly the mastoid cells had been

opened, but nothing had been found. A long clot was removed from the sinus. From that time to April 17 he had done well, but on a return of the symptoms the incision had been extended to the clavicle and the jugular vein opened, no clot being found until the facial vein had been reached. The man had been very ill for several days afterward, and had had a metastatic pneumonia. At no time had there been any tenderness over the mastoid, and no pus had been found in this region. The pain complained of had been almost wholly over the frontal lobe of the brain.

In the light of our present knowledge, cases of thrombo-phlebitis should be divided into three chief types. The first type presents the well known text-book picture marked by sudden chill, high temperature, and abrupt remission with which all of us are familiar. In the second type chills are absent, and there is no high elevation of temperature, one to three degrees, continuing indefinitely without marked remissions, or only gradually returning to normal—the picture of continual drainage of poison into the system. The third type represents the cases with no temperature at all. Tobey found 11 such cases in a series of 73.

The symptoms commonly given as characteristic of sinus thrombosis. In the light of our present knowledge, few, if any, of them are dependable. The following are usually emphasized:

1. *Chills.* The presence of chills will depend upon the amount and the frequency of the release of the poison into the blood stream. Many cases are free from chills throughout the entire course of the disease. In others, all that can be elicited is a doubtful history of chilly sensations.

2. *Headache.* The headache of sinus thrombosis is rarely a severe pain. It is more apt to be a dull ache referred to the affected side. Its presence and severity depend largely upon the type of the disease. This applies also to a condition of drowsiness which is often present. *In type one,* all symptoms may disappear as soon as the patient has recovered from his chill. In the more insidious forms of *types two and three,* headache with other manifestations of sepsis, may be the only symptoms met with, in addition to a slight elevation of temperature.

3. *Knotting in the Neck and Tenderness along the Jugular Vein.* This much mentioned symptom is entirely untrustworthy. It occurs only very late in the disease. Many observers have never met with it.

Eye Symptoms. Contrary to some of the observers who put the changes in the fundus as high as one in three or one in five, most

reporters have found only rarely (not more than 10 per cent or 11 per cent) changes in the fundus, usually in the form of choked disk. When present, these point to a meningeal irritation and infection or an extension of the thrombus. Paralysis of the sixth nerve has been met with by some observers more often.

Blood Culture. The work of Libman at Mt. Sinai Hospital on the blood in sinus thrombosis, is classic. It was hoped for a time that as a result of this work, blood cultures would prove of great help in diagnosis. Time and experience have not, it is to be regretted, borne this out. We now know bacteremia is met with in other pyemic conditions, such as endocarditis and typhoid fever. Further, while Libman's dictum is undoubtedly true that at some stage of the phlebitis, bacteria can be recovered from the blood, it is not always possible to obtain the blood at such times even by repeated examinations. This has led most aurists to the conclusion that positive blood findings, if other causes can be ruled out, are indicative of sinus thrombosis, but not to attach too great importance to a negative blood result.

Edema Over the Emissary Vein. Tobey regards an edema and tenderness over the area drained by the mastoid emissary vein as characteristic of sinus thrombosis. He has found it present practically in every case he has seen. The edema and tenderness never extend beyond the posterior wall of the mastoid. Others have not shared this experience.

Appearance of the Sinus Wall. A change in appearance of the sinus wall in the presence of a thrombosis, is constantly met with. Often the wall presents the so-called characteristic glazy yellow color, the lustre entirely gone. This, however, may lead into error. Crockett[8] has pointed this out, saying that an apparently healthy wall may contain a clot just as a sinus with a presumably diseased wall may be free of all thrombus.

Enlargement of the glands of the neck will be encountered at times, and *absence of pulsation* in the presence of a thrombus, is commonly met with. Finally, even elevation of temperature, which for a long time was regarded as the most dependable symptom, we now know is often lacking.

Quoting Tobey once more: "This great variation demonstrates clearly that the temperature cannot be considered a definite diagnostic symptom. Its presence indicates infection, but its absence or its atypical character do not preclude an infectious thrombosis of the sinus."

Diagnosis. Many of the gentlemen present will testify that no one of these symptoms has been sufficient to enable them, in some

puzzling case suffering from sepsis, to make a diagnosis from other conditions such as central pneumonia and malaria, which at times try to the utmost the skill and ability of the internist. Too often the diagnosis can be cleared up only by exploration of the sinus. Even this in the hemorrhagic type with small or transient clot, or a direct infection of the system without clot, may fail immediately to establish the diagnosis, although, as our honored Chairman[9] has shown, prompt recovery following operation would seem to do this. Finally, in addition to the necessity of making a differential diagnosis from some general systematic trouble, it is often necessary to differentiate between sinus involvement and meningitis or brain abscess. Indeed, it must never be lost sight of that there is nothing to prevent two or more of these affections existing together.

Prognosis. Serious as the condition is, there can be no question that numerous cases of thrombo phlebitis get well. In all probability the number of cases which recover unrecognized and without proper treatment, is considerable, as evidenced by Day's report and others. So far as the outcome of those cases which are diagnosed and properly treated is concerned, much depends upon the nature of the organism and the power of resistance of the patient. In the presence of a fairly sluggish organism, prompt and complete mastoid surgery will cause pyemic changes to subside, and recovery to take place in the great majority of cases. In the presence of a high degree of virulence or of low resisting power, or both, with delayed interference, the outcome is too often fatal. Particularly is early intervention demanded in the hemorrhagic type where direct invasion of the blood stream may take place without apparent parietal clot.

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HAY-FEVER.*

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Hay-fever is a peculiar malady. The persons afflicted with hay-fever are not sufficiently ill to receive flowers, but are quite miserable and willing to accept sympathy. I regret that I am unable to bring forth any new thoughts. I have, however, endeavored to collect data upon the subject and will present it to you in as practical a manner as possible.

The history of hay-fever is as rich in facts as the malady is full with its victims.

Galen and Hypocrates speak about rhinitis with perfuse nasal secretions. They thought that the secretions were an exudate from the brain. It is surprising to know that their thoughts were perpetuated until the early part of the 16th century.

In 1516, Leonhardes Batallus of Pavia speaks of patients who developed asthma from the presence of roses.

In 1565, Botallys (known to fame as the discoverer of the foramen ovale in the heart) affirmed that he knew persons who held the smell of roses in deadly hatred because it gave rise to headaches, sneezing and troublesome itching in the nose.

In 1577, Wan Helmont speaks about a cleric suffering with asthma resulting from the odors of the sweet-smelling substances.

In 1634, Scaliger reports a case of a girl who fainted at the sight of lilies and she felt as though she would die if she smelt them. Schnieder, the Father of Rhinology, relates a case of orange flower which produced syncope.

In 1662, Conrad Victor Schneider, Professor of Wittneburg, speaks of nasal catarrh due to roses. He was the first one to challenge the Hypocrates and Galen theory that the nasal secretions were not exudations from the brain.

In 1673, James Binningerus quotes Professor Brun of the Medical Faculty of the University of Basle. He speaks about his wife who was susceptible to coryza every year for several weeks during the rose season.

In 1684, Samuel Iedelius relates of a merchant who was suffering from nasal itching followed by inflammation of the eyes with profuse lacrimation and who was susceptible to rose fever. The merchant was a close observer of his own ailment and he learned that

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his condition was much better when he remained at home and he was practically free from attack when confined to the house.

In 1691, Jacobus Constant Rebeque described his own condition. He was a victim with rose fever for 13 years.

In 1710, William Heberden reports four patients who suffered with violent sneezing, lacrimation, headaches and itching of the nose. He was the first to call the attention to the fact that rose fever may last during the months of April, May, June and July.

During the period between 1710 and 1819 we find no mention of hay-fever in the literature. Whether this implies self-contentment with the previous theories or pure negligence is not known.

In 1819, however, John Bostock described his own condition and called it a rose cold. He blamed the sunlight and dust as the provocative factor. His description of the symptoms were so perfect that hay-fever established itself as a distinct entity. The history is of extreme importance. It begins on a definite date with a sensation of heat, fullness and redness of the eyes, photo-phobia, itching, smarting, a feeling of lencinating pain in the eyeball, lacrimation which at first is profuse and watery and as time elapses becomes thick, ropy, mucilaginous and a blepharitis results. Sneezing aggravates these symptoms. *Nasal Symptoms:* Fullness in the head, especially over the forehead, tickling sensation in the nose, sneezing at intervals, copious discharge, redness of the nose from manipulation and pain over the ala nasi. *General Symptoms:* Indisposition, languor, loss of appetite, difficulty in swallowing due to nasal secretions, muffled voice, restlessness, increased pulse rate.

From 1856 to 1877, Blakely, who was subject to hay-fever, established definitely that hay-fever was caused by the pollens and that the symptoms became aggravated during the atmospheric changes and that it was more marked when the winds were blowing. He proved this conclusively by using plates smeared with a sticky material, which he examined at different intervals. He found that on windy days there were more pollens and weeds than on calm days. He associated his findings with his own symptoms. His condition was always alleviated when the weather was calm and aggravated during wind storms. This may explain the reason why during the months of June and July, hay-fever is not very prevalent and may be due to the fact that not many pollens are thrown off from the various grasses.

Like all new theories, hay-fever had its setback and its enthusiasts, pro and con. At this time many men began to advance various theories. Some spoke of the nervous theory, others of the atmospheric pressure, sunlight, etc. Then came the great bacteriologic

and microscopic episode with Koch and Pasteur as the shining lights. The enthusiasm and revolution caused by these great men had its influence upon the profession and every one began to seek and attach bacteria as the exclusive causative factor of hay-fever. There were, however, sporadic reports by Cornaz of Switzerland, Labosse of Nitry, France, that hay-fever was caused by the flowers of grasses. It was Morrell Wyman of Cambridge, Mass., who was the first to distinguish the two distinct forms of hay-fever, the autumnal and spring.

About 1882, Daly of Pittsburg, Sajous of Philadelphia, Roe of Rochester and Harrison Allen of Philadelphia, associated the fact that certain nasal pathology may be the soil for the production of hay-fever and that when these deformities are corrected and the pathology removed the condition would be alleviated.

It is surprising to note that as late as 1897 men like Grayson and Edmund W. Holmes of Philadelphia adhered to the theory of neurosis and men like McDonald and Bishop believed that the digestive tract is the cradle of systemic error.

In 1902, the pendulum again swung towards the pollens and Dunbar of Hamburg, Germany, prepared hay-fever antitoxin by inoculating horses with the toxins derived from the pollens. Dunbar thought that hay-fever is a result of a specific poison found in the pollens and it is an albumenoid body; his antitoxin was intended to inhibit or immunize patients against the pollen toxins.

Among the recent workers are men like Oppenheim and Gottleib, Goodale, Walker, and Scheppegrell. They are making this work almost a life study. These men placed hay-fever on the scientific bases as we understand it today. They individually, as well as collectively, worked out the various skin tests for pollens, the preparation of the extracts for the treatment, animal emination and food proteids. They are the ones who fought the gun-shot pollen emulsions.

We know that hay-fever may be divided according to seasons, namely, spring, mid-summer, fall or autumnal, and another called the intermittent group, which occurs independent of season. The latter, Walker calls perennial hay-fever. Hay-fever again may be divided according to the substances producing it, namely, the spring pollens, such as dandelion, June grass, timothy, sweet vernal, red top and rose, which we find as early as May 15. In the mid-summer group we have the orchard grass and daisy, and in the autumnal or fall pollens we have the rag-weed, short and long, sunflower, cockleberry, short and long, alfalfa and goldenrod. The fall hay-

fever is the most prevalent and rag-weed stands as the highest culprit and lasts until the first frost. The other provocative factors are mechanical nasal obstructions, thermal exposure, animal emination, food anaphylaxis and bacterial infection. It is often difficult to differentiate true hay-fever caused by pollenatious substances from vasso-motor rhinitis. The most impressive and definite symptom accompanying hay-fever is the eye syndrome. We have repeatedly noted patients who present themselves with symptoms of hay-fever, not accompanied by conjunctivitis, lacrimation and photophobia. They will invariably yield negative skin tests to the pollens.

Symptoms of Hay-Fever: Nasal congestion, watery secretions, muffled voice due to nasal obstruction, sneezing, general malise and heavy feeling in the head. (Or, as the patient would insinuate, "my head feels as though it weighs a ton.")

Eye Symptoms: Photo-phobia, lacrimation and conjunctivitis.

Pseudo hay-fever or vasso motor rhinitis presents the same symptoms as hay-fever, only minus the eye condition. The provocative factors here may be mechanical irritation, olfactory vassomotor rhinitis, thermal exposure, irritating gases, dust, and last, but not least, face powder.

The preplex problem of hay-fever is assuming a preponderate position in the maladies of the people of this country. One per cent of the total population of this country is afflicted with hay-fever and twenty per cent of these cases develop asthma as a complication. We must therefore consider this problem very seriously.

At present all our energies are centered on pollens and thus far the results are encouraging, but not entirely satisfactory. I believe there must be some underlying factor that kindles the light, as up until our present status of knowledge we have not as yet determined definitely the underlying factors. People may go on until adolescence and middle age and suddenly develop hay-fever. The whys and wherefores we cannot explain, but contenting ourselves with our present knowledge we therefore come back to earth again and speak about the methods of testing patients afflicted with hay-fever.

Two methods at present in vogue are the endermic and the scarification or epidermic method. The endermic method of injecting the pollens is employed and one-tenth of a c. c. is used as a standard dose. The following constitutes a positive reaction: 1. The lesion must persist for more than twenty-four hours, 2. The presence of a distinct papule, 3. The persistence of erythema, 4. Tenderness and induration at the point of injection. The readings are determined as plus 1, plus 2, plus 3, and plus 4. At present the scarification or

epidermic method is most popular. A small area of epithelium is scarified and the pulverized powder is placed upon the area and lightly massaged and in order to enhance the solubility of the protein one-tenth per cent of normal sodium hydroxide is applied. The reaction may be read within one or two hours. When sensitiveness is present a wheel appears with induration varying in size. It is important to note the amount of induration present as we form our conclusions upon this. The causative factor having been determined, treatment is instituted. Walker employs the following formula: The strongest solution that fails to yield the slightest reaction. One-tenth of a c. c. of this solution is inoculated weekly. Caution must be observed before the dose is doubled and trebled so as not to produce any anaphylactic shock. Those of us who are using stock solutions are supplied by the manufacturers with standard dosages and if their rules are closely observed, one need not fear any untoward effects.

Method of Treatment: The treatment is divided into three stages: 1 Before the attack, 2 During the attack, 3 And the intermittent attack. Up until a few years ago, all patients found to be suffering with hay-fever were treated with the pollens, irrespective of time, whether they were free from hay-fever or not. I do not administer pollens to patients suffering with hay-fever unless I begin treatment ten or twelve weeks before the attack and discontinue the inoculations at least four to six weeks before the onset as determined by the history and skin tests. During the onset, vaccines are administered and often the symptoms are alleviated in this manner. Those patients suffering with intermittent hay-fever, pollens and mixed vaccines are used with beneficial results. At the present time we are able to determine definitely what pollens or proteids an individual is susceptible to and in instituting treatment we administer the specific pollen. However, the more one thinks on the subject of hay-fever, the more theories crop up into his mind.

The Endocrines are considered very seriously at present, and there are many facts to substantiate the advocaters of the endocrines. We know that during hay-fever there is a low blood pressure, and an increased pulse rate. We also know that there is immediate immuluration of the symptoms when adrenalin hydrocloride is administered hyperdermically to asthmatic patients. The endo-crynologists are unable to explain what disturbs the functions of the endocreens. I hope with the modern methods of basil metabolism and with the increasing knowledge of physiological chemistry, we will reach further into the unknown

Ernest Sachs, Cushing and Weed, are at present experimenting with hypertonic salt solution for hydrocephalis. They find that the fluids will diminish upon administering this hypertonic salt solution. Taking this finding as a basis, I associate the fact that all hay-fever patients have a low blood pressure. I thought by trying sodium chloride, it would increase diapedesis in other parts of the body, thus withdrawing the congestion from the nasal mucosa; I feel that I have succeeded to a certain extent. I have been in the habit of administering twenty to thirty grains of sodium chloride daily to patients suffering with hay-fever. We are all acquainted with the fact how dry our throat becomes when we eat something salty, and upon administering sodium chloride in the dosage aforementioned, I find that about twenty per cent of the patients develop thirst and complain of dryness in the throat. The sodium chloride acts in a double capacity: 1 It causes dryness of the throat, 2 Dehydration of the tissues. This has an advantage because the patients drink great quantities of water on account of their thirst and thus they neutralize some of the toxins present in the body.

Dr. George W. McKenzie of Philadelphia treats hay-fever in this manner: He collects the nasal secretions upon small pledges of cotton and forces the secretions through the Berkfield filter, which in turn is administered hyperdermically.

I do not remember the exact technique that we used to dissolve some of the substances. In brief it was prepared as follows: By extracting the protein by the use of a weak alkali and, after shaking, the solution was incubated. This was filtered and absolute alcohol was added and the solution evaporated in a water bath. A saturated solution of the dry material was made in an alkalinized sodium chloride solution and the nitrogen content was determined by the Kjeldahl method.

It is very important to obtain a good history. True hay-fever always comes on on a definite date and at certain season of the year, while pseudo hay-fever may come on at any time. I might say that when the cold weather first appears a great many people think they have hay-fever on account of sneezing, and these conditions are often puzzling. Here the history plays an important part and one will invariably learn that some of the winter clothes were taken out of the camphor closet and the camphor dust produced an irritation of the nose.

Face powder is a very important factor. As the powder is placed upon the face carelessly some of it is inhaled, producing an irritation with sneezing. Rhinologic examination reveals a crust formed by the powder and the nasal secretions.

At the present time we do not know as yet whether hay-fever and asthma are caused by the direct irritation of the pollens or whether there is a sensitiveness present to those substances. The pathology that takes place in the nose, reasoned from a purely mechanical standpoint, is as follows: The pollen is inhaled and settles in the nasal mucous membrane. Irritation sets in with congestion of the mucous membrane and dilation of the lymphatics. The function of the cilia are disturbed and none of the substances are readily thrown off. A chemical change of these proteids takes place in the nasal chamber and absorption of these noxious substances results and the complicating symptoms which follow I believe are the result of this phenomena. How far the endocrines play a part we do not know. I suppose time will show. I never promise any one afflicted with hay-fever what the results will be after treatment. They do, however, respond better in spring and mid-summer.

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COMMUNICATION.

Editor of THE LARYNGOSCOPE:

Apropos of Dr. William Spielberg's article in the June issue of THE LARYNGOSCOPE regarding his employment of Holmes pharyncoscope to inspect the interior of the maxillary sinus through an opening below the inferior turbinate, I wish to say that this method has been employed at the Hajek Clinic in Vienna for some time. But instead of making this a routine method of examination of every suspected case of disease of the antrum, it is used mostly after performing the Mikulitz operation or its modification.

When I went to Europe last fall, the Holmes pharyngoscope was one of the few articles from my instrument case that found their way into my trunk, and I made good use of this instrument in examining a number of maxillary sinuses at the Hajek Clinic.

Halle of Berlin employs the Canfield method of examining the maxillary sinus, and I believe this is far superior to the method described by Dr. Spielberg. It consists of cocainizing the inferior and middle nasal passages, anterior portion of inferior turbinate and corresponding part of the septum, by the application of 20 per cent sol. cocaine with equal parts of adrenalin solution. A 1 per

cent novocaine adrenalin solution is then injected subperiosteally into the canine fossa through the nose.

An incision is then made from the middle of the anterior attachment of the inferior turbinate extending to the floor of the nose, through the soft tissues down to the bone. With a Halle or Freer elevator the periostium is elevated from the crista pyriformis, and about ½ inch externally toward the canine fossa. With an electric trephine the antrum is now opened. It takes a few seconds to go through the thin facial wall. The opening can be made large enough to admit a moderate size ear speculum or, better still, the electric auroscope, through which the interior of the antrum can be inspected.

If the sinus is normal, a stitch or two will close the opening in the soft parts and no harm is done, as the bony opening will close in a short time.

If I find the antrum to contain pus, I irrigate it with boric acid solution until the return flow is clear. Then with a metal tube similar to the Eustachian catheter, but shorter, I aspirate the remaining fluid and inspect the interior of the sinus with the electric auroscope. If no ploypi are present and if the mucous membrane is in a fairly good condition, a few daily irrigations will suffice to clear up the condition. The opening in the anterior wall closes up as rapidly as the one under the inferior turbinate. If, however, polypi are present, if the mucous membrane is found degenerated in spots, or areas of granulations are present, a more radical operation is indicated. This can be performed by enlarging the opening in the facial wall of the antrum, removing part of the crista pyriformis and part of the nasal wall below the inferior turbinate. This part of the inner wall of the antrum is removed from within the maxillary sinus, so that the nasal mucous membrane covering it is left intact. An inverted U-shaped incision is then made in the mucous membrane below the inferior turbinate and the flap turned into the maxillary sinus on its floor. The antrum is packed with iodoform gauze through the opening below the inferior turbinate so as to hold the flap in position. The facial wall is removed with an electric trephine while the inner wall is removed with Hajek bone-cutting forceps.

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SUCTION TREATMENT IN ACUTE INFECTIONS OF THE EAR.

Dr. E. R. Carpenter, Dallas, Texas.

A great deal has been said and written about acute infections of the ear, and yet with the exception of a free paracentesis when indicated, but very little has been accomplished in the way of actually controlling the situation. The ear is often irrigated, various antiseptic drops are used, heat and cold are applied and along with natures efforts many cases make a good recovery. However, certain cases steadily increase in severity until well pronounced symptoms of mastoiditis are evident, either within the first few days, or at some later period. Often a chronic suppurative otitis supervenes without apparent mastoid involvement. This is especially likely to occur with the infectious diseases of childhood. Probably the majority of all acute suppurative ears have more or less mastoid involvement without pronounced symptoms.

From careful observation of these acute conditions, under a somewhat heroic method of treatment, I am convinced that ninety per cent of the usual acute mastoid operations can be avoided and that chronic suppurative otitis media should rarely ever occur.

The treatment under consideration applies to all cases where a paracentesis should be made, or where the ear has ruptured spontaneously early in the trouble. The etiology is immaterial beyond the fact that adenoids should be removed when present in any child that has ear complaint, and that syphilis sometimes causes acute ear trouble.

When a paracentesis is indicated it should be done under some kind of anesthesia and as large an incision should be made as is consistent. Then immediately apply suction by means of an ear tip large enough to completely close the external canal, allowing the blood and pus to drain into the bulb of the ear piece or other connecting receptacle. From five to fifteen pounds or more pressure can be used safely. I have never seen any damage from suction in this form, but it should be used more or less intermittently. I sometimes remove half an ounce or more of bloody fluid. The canal is then gently cleared of clotted blood and several drops of two to five per cent mercurochrom solution are instilled into the canal and with the suction apparatus this is gently pressed back into the middle ear by alternate suction and compression, using the same ear tip for this

purpose. Potassium mercuric iodide is also very good in these cases and is less troublesome than the mercurochrom solution.

Some of the hand suction outfits have sufficient suction power to remove the free blood and relieve the stasis, but the Robertson office outfit is the ideal apparatus for this work, as various combinations of suction and compression can be obtained with it in addition to either suction or compression.

The ear is kept lightly packed with a gauze wick and the suction is used two to four times a day as long as needed. Before each subsequent treatment is begun, two or three drops of a ten per cent cocain solution should be instilled into the canal and gently compressed into the middle ear. There is some discomfort during the treatment, but not excrutiating pain. Rarely a general anesthesia is advisable in the after treatment. Where practical the physician should carry out the method, as he soon acquires a technic that accomplishes far more than when used halfheartedly by the inexperienced; however, at times it is necessary to instruct a nurse or other attendent to carry out the procedure as best they can at home with a hand suction outfit.

Naturally less suction or more careful handling for babies is required than for older children and adults. As a rule I never use irrigation or other drops than the mercury solution. Aside from removing secretion from the middle ear, antrum and mastoid and overcoming stasis, this treatment keeps the incision open during the treatment and the wound closes nicely after recovery. The earlier the treatment is instituted, the better and quicker the recovery. Treatments should be continued until the secretion has about ceased. The number of treatments vary as to the case, the average case of otitis media is greatly improved within a few hours and often only two or three treatments are necessary. Where the antrum and mastoid are involved early in the process more persistent treatment is indicated and may require several days of unrelenting care. Occasionally when a case is seen rather late and mastoiditis is evident, the mastoid and antrum may be entirely closed off from the middle ear by severe swelling of the mucous membrane, such rare cases are usually operative and are recognized by the unsatisfactory response to treatment and by the limited amount of secretion that can be removed by suction. As a rule the treatment is not advocated when mastoiditis develops after the acute otitis has persisted for two weeks or more, as here the possibility of extensive necrosis must be considered.

SUMMARY.

1. The method has been used in many cases with no untoward results.

2. The course of acute infections of the ear is cut short by the proper use of suction and compression and the treatment usually prevents complications.

3. Results depend much on the thoroughness with which the treatment is carried out.

4. The treatment is often valuable in other than acute conditions and is especially useful in assisting nature to remove secretion from the middle ear and to eliminate stasis during the after treatment of acute mastoid operations.

5. Altho other forms of treatment are not deemed necessary, yet they may be used in connection with suction if desired. (Especially heat and cold.)

6. No claims are made for priority in the use of suction in ear work, but the method I have described has enabled me to treat these acute infections much more successfully than by any other means with which I am familiar, and without reservations I recommend its use.

ILLUSTRATIVE CASES.

Case 1, J. J. age 8, had earache one week before family physician referred him to me. Child cried incessantly, mastoid was very tender, drum was dark red and bulging. He had high temperature and rapid pulse. General anesthesia was administered at home, drum was incised and two or three drams of pus and blood were removed by hand suction outfit and mercurochrom was instilled. He was much relieved within a few hours. Two or three subsequent treatments were given when the secretion consisted only of blood. Recovery within a few days was uneventful except that the other ear became involved within forty-eight hours after I first saw him. Free incision of the drum membrane and one treatment with the suction outfit permanently relieved this disturbance.

Case 2, A. S. W. age 52, caught cold three weeks previously and left ear became infected one week before I saw him. He had suffered much, the left mastoid was very tender and edematous. The drum membrane was under considerable pressure, he had some fever and rapid pulse and felt very badly. Local anesthesia was used in the office, the drum was incised, and three or four drams of blood and pus were removed by suction. He was relieved within a few hours, treatments were continued several days or as long as any pus could be obtained. The incision closed promptly after about two weeks observation. The mastoid and antrum could be drained freely in this case by suction.

Case 3, J. H. L. age 21. This man never had any ear trouble until three weeks before I saw him, at which time he caught cold and his right ear became infected and he had suffered much for a week from mastoid pain. Examination revealed all the symptoms of an acute mastoid infection, but no pus was draining from the middle ear, altho the drum had ruptured several days before he came to me. General anesthesia was administered and the drum was incised, but only clear blood could be removed by suction. An acute mastoid operation was made at once. The mastoid cavity was blocked off from the middle ear by severe swelling of the mucus membrane, much pus and necrosis was present. The wound was closed with the exception of a small rubber tube drainage at the lower end of the incision. Middle ear and antrum were cleared of all secretion and blood at the time of the operation by suction, which was repeated daily for several days. Patient made an uneventful recovery and returned to his work in a distant town in ten days. Suction treatment after the operation assisted materially in rapid recovery.

Case 4. A boy, eight years old, and the son of a physician, caught cold and developed otitis media; the drum was opened by a small incision and suction used, but insufficient to get results; fever continued high, tenderness of the mastoid developed. In two or three days I saw the case and under gas anesthesia I made a large opening in the drum and used suction freely, as much secretion came away. He improved at once, but in two days fever recurred with renewed tenderness and swelling of the mastoid. I was called again and repeated the treatment with equally as good results as before. After an interval of three days I was called and found the child in a serious condition. Drainage was poor and suction had been used in a half-hearted way. Again I repeated the treatment and returned two or three times daily for three days and used suction energetically. He improved at once and made a rapid recovery.

NOTES ON PERORAL ENDOSCOPY AND LARYNGEAL SURGERY.

DR. CHEVALIER JACKSON, Philadelphia, Pa.

Bronchoscopy in Asthma and "Asthmatic Bronchitis." It does not seem unreasonable to expect that bronchoscopy will eventually bring the same revolutionary results in the diagnosis and treatment of pulmonary disease that the development of local methods of examination and treatment has brought to every locally reachable organ in the body. Notwithstanding this justifiable expectation, it is true that the progress so far has been slow. Apparently the slowness is due to the fact that bronchoscopy is a sort of appendage to the enormous and more attractive field of otology and rhinology, a status which has prevented the development of facile work in bronchoscopy. To pass the bronchoscopy is looked upon by the occasional bronchoscopist as a formidable undertaking; whereas in reality it is, technically, a mere trifle as compared to, for instance, the cystoscopic work necessary for the modern study of renal disease. A few very interesting articles dealing with bronchoscopy in disease have appeared. One by Gottlieb* on the endobronchial treatment of asthma is particularly worthy of study, as will appear from the following quotations: "An idea has been assumed by the great majority of physicians that, given a case of bronchial asthma that is found sensitive to one or more proteins and having eliminated these from his economy and environment, the symptoms should disappear. This is undoubtedly true if the case is not complicated by chronic respiratory infection. Most of the cases have in addition a chronic bronchitis and that is the reason why failure to cure occurs in so many cases. A generalized infection of the bronchial tree with peribronchial changes with or without allergy is, so far as my experience goes, almost hopeless of relief or cure, as was shown in two cases. Factors that are not usually considered in the etiology of bronchial asthma, which to my mind are very important, are susceptibility of the bronchial mucous membrane to reinfection, habit and the psychology of the patient, as was demonstrated in three cases. Whenever they acquired acute nasal infection it was always followed by infection of the bronchi with an aggravation or return of their asthmatic symptoms. In such cases surely the bronchial mucous membrane is a locus minoris resistentia. The constriction of the main bronchus in these cases is either due to a swelling of the mucous membrane or

*Gottlieb, Mark J. The Endobronchial Treatment of Bronchial Asthma and Asthmatic Bronchitis. Journ. Amer. Med. Assn.

a spasm of the muscular coat of the bronchus which lies directly underneath the mucosa, or a combination of both. Whether the spasm of the bronchial musculature is due to direct irritation at the site of infection or to reflex spasm from the upper respiratory tract, it seems quite reasonable to suppose that one or both factors may be operative. It is a very well known fact that nerve pathways that are traversed constantly transmit impulses more readily and with greater facility than those which are occasionally used. This is very well demonstrated in piano playing and in other manual habit-formed occupations. This may also apply to patients with bronchial asthma who suffer with a recurrence of their symptoms while undergoing mental distress or following an infection in some other part of the body, particularly in the upper respiratory tract. With these considerations in mind, it is necessary that whenever possible patients should be free from worry and aggravation. They should be instructed to avoid crowded places and especially to abstain from coming in contact with people who are suffering from respiratory infections. If their living quarters are poorly ventilated and devoid of sunshine, they ought to be changed. Vaccines of the predominant organisms found in the sputum or nasal discharges are of value, especially in those cases in which the individual is sensitive to autogenous suspensions of these, with the exception of bacillus influenza. As was seen in three of the cases, vaccines of influenza bacilli had not the slightest effect in ameliorating the symptoms. Endobronchial treatment is the most valuable procedure that we have at our disposal for treating cases of asthmatic bronchitis and bronchial asthma when the elimination of the allergic substances and vaccine therapy combined with the other measures suggested have failed to produce the results desired. I am firmly convinced that astringents such as silver nitrate and tannic acid have not only the property of temporarily dilating the lumen of the bronchial tree, but also when applied often enough and at short intervals, say weekly, the effect of rendering the mucous membrane less susceptible to infection."

Syme* in a very important and interesting article reports encouraging results of treatment of asthma by the bronchoscopic application of a 10 per cent solution of argentic nitrate, in twenty-three patients ranging from 10 to 60 years of age. One of the patients had four applications, four had two applications and in eighteen only a single application was made. In twelve there was a relative cure, no attack of annoying severity having occurred. Two were unimproved. Nine had varying degrees of improvement.

*Syme, W. S. Bronchoscopy in the Treatment of Asthma. Journ. Laryngol. and Otol., Sept., 1921, p. 427.

Realizing fully the fact that endobronchial treatment is adding another to an already enormous list of remedial measures for asthma, nevertheless the reviewer feels that the wonderful results obtained by local examination and treatment of other regions justify an optimism that warrants the earnest endeavor of every bronchoscopist.

Arachidic Bronchitis. The reaction set up by the peanut kernel has been carefully and accurately observed by Chamberlin*, who reports a very interesting case in a child, aged 4 years, in whom he removed the peanut kernel without anesthesia. Murphy, in two very interesting articles* reports, among other very interesting cases, 5 instances of peanut kernels in the bronchi and one case of field corn (maize). In all of the cases there was a characteristic reaction, the bronchi being in a severe state of inflammation and containing a thick, tenacious mucus, which added to the difficulties of bronchoscopy. The asthmatoid wheeze was present in three of the cases. In all of the cases peanut kernels the fragment of peanut was removed by bronchoscopy without anesthesia, general or local, and the children all made complete recoveries. In the case of the grain of corn (maize) a number of pieces of broken grains were removed aggregating in size about a half grain of corn. The condition of the child did not improve and the next day it died apparently in collapse. A hasty tracheotomy was done and the trachea was found filled with purulent secretions and it contained another grain of corn, the latter being evidently the direct cause of death. An additional peanut case is mentioned in which there was the history of the child having choked while eating peanut candy, but as there were no symptoms or physical signs to indicate the presence of an irritating foreign body in the lung a bronchoscopy was not done. The child completely recovered. The author rightly remarks that we should not bronchoscope every case with a foreign body history without the corroboration of some symptoms or physical signs.

H. L. Lynah reports* a fatal case of arachidic bronchitis from the aspiration of portions of a peanut kernel by a child aged three years. A marked asthmatoid wheeze, expiratory dyspnoea, and diffuse bronchitis, immediately followed. After bronchoscopic removal of portions of peanut kernel, the lung condition failed to improve, although no further portions of peanut could be found on bronchoscopy on the following day. A small fragment had lodged out of the

*Chamberlin, William B. Difficulties Encountered in Removing a Peanut from a Bronchus. "The Laryngoscope," Nov., 1920.

*Murphy, John W. Foreign Bodies in the Bronchi of Very Young Children, Trans. Amer. Laryn., Rhin. and Otol. Society, 1919, and Report of the Removal of Foreign Bodies from the Bronchi and Esophagus, "The Laryngoscope," Feb., 1919.

*Medical Record, February 7, 1920, Vol. 97, p. 215.

line of vision in the dorsal branch of the lower lobe bronchus. Pulmonary abscess developed and ruptured into the pleura. This was drained by rib resection, but death occurred six weeks after the accident, due to septicemia and the specific toxemia of the peanut kernel.

Benign Stenosis of the Esophagus. Green reviews* different methods of treatment of esophageal stricture, including the string swallowing, string sawing and retrograde dilatation methods of Dunham, Abbe, Mixter and others in the pre-esophagoscopic days. These methods, as Green states, still have a sphere of usefulness. The statement, "It is very difficult even by means of an endoscope to locate the cardiac end of the esophagus from within the stomach," seems a little too strong. It is a procedure that often requires patience and is not so easy as might be anticipated, but it can always be accomplished by patient search, if a proper form of retrograde esophagoscope be used. It has advantages over the peroral route in some cases that make it well worth the trouble. As pointed out many years ago by Mixter, there is pouching above whereas there is a funnel-like narrowing below. Green summarizes the results in his eight cases as follows:

In all there were eight cases. Two were adults; six were children three years old and under. One adult stenosis was due to sulphuric acid. One was recurrent after being treated in childhood. One was without history of burn, and was unquestionably congenital. It was coupled with stenosis of the outlet of the stomach. One in an infant gave the history of coming on after diphtheria. All eight are improved. All but one are cured with the necessity of occasional bouginage. One requires at the present writing further slight dilatation by the oral route. Four had the string-sawing operation of Abbe. These were the worst cases and have done very well. Four had dilatation through the esophagoscope and were not treated so vigorously. The results are good, but all kept track of are dependent upon following up with the bougie. The longest interval in the last six cases in which the bougie has not been passed is a year. In the milder types of stricture dilatation through the esophagoscope is sufficient. In the severer types the string-sawing operation of Abbe is indicated.

Diagnosis of Foreign Bodies in the Lungs. In looking over medical literature one is impressed with the fact that foreign bodies in the lungs, if mentioned at all, are regarded only as curiosities of medicine, not as everyday diagnostic possibilities for exclusion in every case of acute or chronic chest disease. The advent of the Roentgen ray and the bronchoscope have revealed so many cases of

*Annals of Surgery, June, 1921.

what was formerly a rarely discovered condition that the leading clinicians are constantly discovering foreign bodies on a tentative diagnosis. McCrae reports a case in which, in the absence of a foreign body history, a foreign body was suspected in a case of pulmonary abscess. A Roentgenray examination revealed a tooth in the lung. One of the leading cases of this kind is that of George L. Richards*, in which an unexplained leucocytosis along with the intermittent coughing up of a small amount of pus led him to place foreign body as the first diagnostic possibility for exclusion. A Roentgenray examination revealed a tack in the lung. The patient, an intelligent man of 25 years, never remembered having had a tack in his mouth. Two important articles indicate that the clinician of today fully realizes the necessity of considering foreign body in all cases of acute and chronic chest disease. One of these articles, by Thomas McCrae*, considers the matter from the viewpoint of the physical diagnostician. The article should be studied. McCrae summarizes his conclusions as follows:

"1. Cases of foreign body in a bronchus are not mere curiosities, but are more common than we have supposed.

"2. There may be no disturbance at the time of entrance of a foreign body and no suggestion in the history of such a happening.

"3. Certain signs are of value, especially decreased expansion on the affected side, the presence of very fine rales and the 'asthmatoid wheeze.'

"4. Some foreign bodies, such as a peanut, set up a very acute general process which is fairly distinctive. Other structures, such as metallic objects, cause permanent changes, usually in a lower lobe.

"5. The chief errors in diagnosis are to mistake the signs for those of pneumonia in the early stages and in the acute cases, and for tuberculosis after the body has been present for some time."

Edwin E. Graham* presents from the viewpoint of the pediatrist a very interesting study of the symptomatology and diagnosis of this subject. His conclusions are as follows:

CONCLUSIONS.

1. It seems reasonable to believe that foreign bodies in the air and food passages in children are much more common than was formerly supposed. Statistics tend to show that about 66 per cent of these cases of foreign bodies in the air passages occur in children.

*Proceedings American Laryngological Association, 1915.

*McCrae, Thomas. The Physical Signs of Foreign Bodies in the Bronchi. Am. Journ. Med. Sciences, March, 1920, No. 3, Vol. CLIX, p. 313.

*Graham, Edwin E. Foreign Bodies in the Air and Food Passages. President's Address. Am. Pediatic Society. Am. Journ. Dis. of Children. Feb. 1920, pp. 119-130.

2. The period of latency of symptoms which follows the violent dyspnea and choking attack, and later the gradual onset, and chronic character of the symptoms, might lead one to fail to suspect the presence of a foreign body.

3. Foreign bodies are certainly often overlooked, as a study of the histories of many cases clearly shows.

4. The symptoms vary greatly. The peanut kernel immediately sets up a severe laryngitis, tracheitis and bronchitis. In the peanut cases, the older child may survive the acute symptoms, but almost surely will develop pneumonia.

5. Metal objects may remain in the lung for a very long while and do comparatively very little damage.

6. There are some foreign bodies that do not cast a shadow on the plate.

7. The location of a foreign body in the esophagus that does not cast a shadow on the plate may often be diagnosed by allowing the patient to swallow a bismuth filled capsule. The Roentgen ray shows the location of the bismuth capsule held in position in the esophagus by the foreign body.

8. Do not urge the patient to cough with the hope of expelling the foreign body inhaled in the lungs.

9. Foreign bodies are very rarely coughed up.

10. The physical signs and symptoms vary according to the composition, form, shape and size of the foreign body.

11. One should suspect a foreign body if the following conditions are present: an unexplained leukocytosis, localized symptoms in one lung that do not clear up under treatment, no tubercle bacilli in the sputum, and a gradual failure in weight and strength.

12. There are no contraindications to bronchoscopy except, perhaps, extreme weakness in the patient, when time should be given the individual to rally partially.

13. Bronchoscopy should be performed as soon as possible after the entrance of the foreign body.

14. Children do not require the administration of an anesthetic for the performing of a bronchoscopy.

15. The necessity of taking a Roentgenogram in every case with a history of swallowing or inhaling a foreign body, cannot be emphasized too strongly.

16. The asthmatoid wheeze, when present, is a sign of considerable importance.

Sand Spurs in the air passages. In a very interesting article Taylor* reports eighteen cases of sand spur occurring as foreign bodies in the air passages; all but one of the cases were in children. In 16 cases the spur was arrested in the larynx, whence it was removed by the direct laryngoscope. In 2 cases the spur was in the bronchi and was removed with the 5 mm. bronchoscope. The author accounts for the frequency of the sand spur in his experience by the great prevalence of the sand spur bearing grass in Florida. The other eleologic factor he describes as follows: "The child notices the sand spur sticking to some part of his clothing or perhaps to his bare feet. With his fingers he attempts to remove it. The bur pricks his fingers, and, child-like, when in pain, he carries his hand to his mouth; the sharp spines of the bur penetrate the highly sensitive mucosa of the tongue, causing a quick cry. The sequel of the cry is a deep inspiration, and the sand spur is drawn in."

*Taylor, H. Marshal: Endoscopic Removal of Sand Spurs from Larynx and Tracheobronchial Tree. Journ. Am. Med. Assn., August 27, 1921, Vol. 77, p. 685.

TOTAL BLINDNESS OF BOTH EYES IN A BOY SEVEN YEARS OF AGE CURED BY ETHMOID OPERATION, AND OPENING SPHENOID SINUS.

DR. DAVID N. HUSIK, Philadelphia, Pa.

Total blindness, secondary to sinus disease, in children is a rare enough condition to warrant my reporting this interesting case.

W. B., 7, referred by Dr. Geo. E. de Schweinitz on March 13, 1922, for nose, throat, and sinus examination to see if there is any foci of infection to account for his sudden blindness.

History of Present Illness. Was perfectly well till February 20, when he developed a severe cold in his head, with marked rhinorrhea, and some frontal headache, but no fever. The cold persisted for two weeks, with hoarseness, and cough. On March 3, patient began to complain of difficulty in seeing objects, which became worse, followed by periods when he could see well. The impairment gradually became worse, and his parents brought him to the University Hospital on March 11, to see Dr. de Schweinitz. He could distinguish colors, and name objects when placed in front of

his eyes; drops were instilled, and was instructed to report in two days.

Examination March 13 shows him to be totally blind in both eyes, and it was at this time that the case was referred to me for examination.

Nasal examination: Septum in the medium line, inferior turbinates slightly injected, but free from septum, and nasal floor. Middle turbinates injected, and in contact with outer nasal wall. There was no exudate visible in either of nasal chambers. Transillumination negative.

Naso-pharynx: Full of adenoid tissue, faucial tonsils slightly enlarged, and cheesy material could be expressed from their crypts. Larynx negative.

On account of the seriousness of his condition, in spite of the negative clinical examination, I suggested the removal of the major portion of the middle turbinated bodies, curetement of ethmoids and opening of sphenoids on both sides, to which Dr. de Schweinitz agreed. No X-ray plates were obtained.

Operation March 14, University Hospital, general ether anesthesia. The middle turbinates were plastered with a 4 per cent cocaine in 1-1000 adrenaline solution for 10 minutes. The anterior half of both middle turbinates were removed, and the remaining portions were fractured over towards the septal side. The bulla was opened, and the ethmoids were removed with curet and Grunwald forceps. The fractured portion of middle turbinates were brought back to the outer wall, and the sphenoid sinuses were opened with a Grayson burr. No pus or a suggestion of pus was seen at the time of operation. Tonsils and adenoids were removed at the same time, the anterior portion of the nares were lightly packed with sterile gauze, and patient returned to bed in good condition.

That night patient bled quite freely, temperature 102.5, pulse 120, and complained of a severe headache.

March 15: Packing removed, temperature 102, pulse 120, headache somewhat relieved, and can distinguish dark from light.

March 16: Muco-purulent discharge from both nares, temperature 100, pulse 100, could see fingers in front of his eyes, but still complains of headaches.

March 17: Temperature and pulse normal, headaches very much relieved, and could count fingers in front of his eyes. There is a profuse purulent nasal discharge.

March 18: Could count fingers at a distance of five inches, headaches all gone, is sitting up in bed, and is feeling fine. His nose is

cleansed daily, and from this day on he gradually improved, and was discharged from the hospital April 1.

Patient was instructed to report every other day for nasal treatment, and during the month of April had several acute attacks of rhinitis, which had no effect on his eyes.

Wassermann reaction negative.

Eye examination by Dr. T. B. Hollaway May 12, O. D. 6/9, O. S. 6/12.

Examination by Dr. B. F. Baer, Jr., June 2. The vision in the right eye is 6/6, and in the left eye 6/6. He can with either eye read the smallest diamond type. The visual fields are full, and there is no central color scotoma. The eye grounds show optic nerve-heads of much paler color than normal, with some filling in of the physiological excavations. The retinal arteries are possibly slightly reduced in calibre, and show some white streaking in the region of the nerve-heads. The eye grounds are otherwise healthy.

Sudden blindness is either due to a toxic neuritis, or pressure neuritis. If due to a toxic neuritis the toxins may come from teeth, sinuses, tonsils, kidneys, etc. If due to a pressure neuritis, it usually comes from pressure in the sphenoid sinus.

In sudden blindness, when teeth, tonsils, etc., are negative, and in spite of negative clinical nasal and X-ray findings, I think we are justified in at least uncapping the ethmoids and opening sphenoid to relieve pressure. The earlier the operation the better the result, and there is no doubt that the above case was one of retro-bulbur neuritis, secondary to pressure in the sphenoids.

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SENSITIZATION DISEASES.*

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It has become a fundamental biological truth, that the entrance of an undigested foreign protein, formed or unformed, into an animal produces profound chemical and immunological changes. The substances injected have been called antigens and the chemical biological changes have been collectively called antibodies. The number and exact nature of antibodies are only recognizable by the reactions they produce and nothing is known of their chemical structure or how they produce reactions. The mere demonstration of these antibodies in the circulatory blood is but one phase of the changes that have taken place in the treated animals and is no index to the intracellular reaction. All tissue cells suffer the most profound changes in the manufacture of immune bodies and upon this fact ultimately depend the explanation of the physiological reaction when an antigen and its antibodies are combined. This physiological cellular phenonema is a protective mechanism for the more effective assimilation and elimination of the foreign proteins that have entered and the mechanism is different when cellular and soluable proteins are injected. [1]Magendie in 1839 and [2]Richet in 1902 found that the first dose of a foreign protein injected into an animal was followed by an increased susceptibility for the specific protein. The syndrome of symptoms which follows the union of the specific antibodies which are produced by a previous dose of protein and the antigen in an animal, results in shock or injury which only occur following the reinjection of the protein after a properly spaced interval. This phenonema is called anaphylaxis, hypersusceptibility, or better, allergy. If hypersensitiveness to other substances as chemicals and not protein solutions are included, allergy is the better term.

Sensitivity may be produced by injecting proteins of either animal or vegetable origin. The condition is specific for the protein injected and the hypersusceptibility develops only after the lapse of a definite interval, usually about twelve days. The interval varies to a certain extent as to the amount administered. An animal once sensitized may remain so for a long period. The condition of sensitiveness can be transmitted from one animal to another by the transfusion of blood. The sensitiveness can be made to disappear for

*Read before Section on Rhinology and Laryngology, N. Y. Acad. of Medicine, Jan. 25, 1922.

a short period of time immediately after the recovery from a non fatal dose of protein. This period is called period of desensitization or antianaphylaxis and upon it is based the rationale of treatment in anaphylactic diseases.

The exact nature of the toxemia produced when an apparently harmless protein is injected perenteally into a sensitized animal has not been proven. Freidberger thought anaphylatic shock due to destruction of the blood complement. Vaughan[3], Jobling[4], Peterson and I have observed the presence of toxic protein in blood following anaphylaxis which is considered an etiological factor in the production of shock. Further [4]Jobling, Peterson and I have observed decided blood ferment and antiferment changes during anaphylactic shock in dogs. In a recent publication[5] I have shown by experiments upon rabbits, dogs and guinea pigs a decided reduction in the alkali reserve of the blood immediately and before the advent of other clinical symptoms of shock. Zinzer and Porges think anaphylactic shock results from a disturbance of the protective blood colloids. All these theoretical explanations fail to entirely explain this complex and violent reaction. Anyone experienced in experimental production of anaphylaxis in animals is impressed with the profound symptomatology produced by the reinjection of foreign protein into a sensitized animal.

For a long time anaphylaxis has been considered an acute phenonema and only recently have milder allergic reactions in animals received detailed investigation and by these experiments there has developed an application of the phenonema to practical medicine. The relation of sensitization to many diseases entities has received its greatest impetus in the last decade. Notably is the work by [6]Von Pirquet upon tuberculin, [7]Blackley, [8]Schloss, [9]Cooke, [11]Selfridge, [12]Kessler, [13]Goodale, [14]Scheppegrel and others upon hay-fever, [6]Von Pirquet, [7]Blackley, [8]Schloss, [9]Cooke, [10]Walker, [11]Selfridge, [12]Kessler, [13]Goodale, [14]Scheppegrel, asthma, chronic bronchitis, eczema and urticaria.

Diseases now classed as some phase of allergy are:

First: Serum disease. Second: Hay-fever; seasonal and perennial. Third: Asthma; Food, Emanations and powders, Bacteria. Fourth: Skin: Urticaria, Eczema, Acne. Fifth: Conjunctivitis; Acute, Chronic. Sixth: Acute gastric collapse. Seventh: Acute gastroenteritis, non infective. Eighth: Migraine.

Serum disease results frequently after the administration of horse serum in the treatment of hemorrhage, diptheria and pneumonia. Large numbers of patients are naturally sensitive or have acquired

sensitiveness to horse protein. The sensitiveness may be due either to the previous injections of serum or by the absorption or inhalation of horse dander and emanations thru the mucus membrane of the nose or conjunctiva. This disease has become relatively frequent since serums are being used in the prevention and treatment of diseases and particularly in pneumonia where large doses of serum are administered. No patient should receive a dose of foreign serum without the physician having first determined if he is sensitive. This can easily be ascertained by intracutaneous injection of 0.1 minim of the protein to be used. A reaction will appear within half an hour if the patient is sensitive, and consists of an urticarial wheal surrounded with an area of erythema. If the reaction does appear small doses may be administered repeatedly with extreme caution until large doses can be given.

[15]MacKenzie recently published a routine to follow in desensitizing a patient. If no reaction occurs large doses of foreign protein may be given with impunity. The preliminary test upon each patient to whom serum is given is absolutely indicated. Quite a number of deaths have occurred from failure to observe this precaution and the number of deaths will increase as more foreign sera are used. One cannot depend upon a history of the previous administration of a serum to eliminate the possibility of hypersensitiveness because this may be a natural state or the patient has acquired hypersusceptibility as stated to animal emanations. The symptoms of serum diseases may be severe unto death or mild with slight urticarial rash, edema with severe itching and burning rendering the patient most uncomfortable. In serious cases a low blood pressure and dyspnea occur with purpura, cynosis, asthma, convulsions and death.

HAY-FEVER.

Seasonable hay-fever has been definitely grouped as an allergic disease and is produced by a sensitization to pollen proteins. The disease is manifest therefore when a patient receives a second dose of pollens which occurs only when the plants pollinate. Blackley in 1865 made the first skin test to pollens for hay fever, but could not progress because little was known of immune reactions at that time. [12]Koessler 1913, contributed much to our present day development of pollen therapy of hay-fever. The work of Goodale, Selfridge, Scheppegrell, Walker and Cook have shown the relationship of pollens to the seasonable types of hay-fever. Cooke and [16]Coca were unable to prove this relationship to be a form of anaphylaxis, however, [17]Parker has recently shown that this relationship is in the form of anaphylactic shock. Hay-fever is not a local disease

of the ear, nose and throat, it is a general condition and operations upon the nose will not cure anaphylactic hay-fever. Well advised operations will give relief in those cases where there is a nasal obstruction and poor breathing space. In these cases operation is always advised but the operation will not alter the general condition of pollen sensitization.

The symptoms of hay-fever are usually manifested in the eye, ear, nose and lungs, because the pollen dusts fall upon susceptible mucus surfaces and does not effect the skin which is impervious. However, itching, redness, swelling can be produced in an open skin wound upon any part of the body if the pollens come in contact with the wound, proving the disease is a general condition and not a local disease. This principle is used in determining which pollen is responsible in each individual case. Hay-fever victims manifest general symptoms as nervousness, irritability, depression and weakness.

Pollinosis may be of the spring, summer and fall type. In the early spring it is usually a manifestation of the sensitization to the tree pollens. Pollens of many varieties of trees, grasses and plants are necessary for testing patients in order to find which is responsible. In some cases as many as fifty varieties of pollens are used before the causative one is found. When the patient has been tested, and the pollen to which he is sensitive is found, the patient is desensitized. If several pollens are responsible for the disease they are combined in the proportion to which the patient reacted, and the mixture is used in treating the case. Hay-fever during May, June and July has incorrectly been called rose cold. Roses rarely cause this condition as rose pollens are heavy and cannot be as easily transported by the wind, this is not true of the pollens of red top, timothy, orchard grass, june-grass and sheep fiscus. These are the ones usually responsible for the early summer hay-fever. The August and September types of hay-fever have popularly been accredited to goldenrod pollens but this plant has been unjustly accused in the majority of cases. It has been found by testing a large number of patients that 90 per cent of the autumnal hay-fevers are due to ragweed pollens. Corn pollens are quite commonly the cause of hay-fever in farmers.

In the hay-fever clinic at The Manhattan Eye, Ear and Throat Hospital, routine tests are made for each patient at the beginning of his case using the pollens as suggested by the seasonal history of the patient. Tests are made by the cutaneous or intracutaneous application of the suspected pollen. The reaction produced, if the

patient is sensitive, appears in about 10-20 minutes and consists of an ameboid urticarial wheal and an adjacent zone of erythema. When a patient has been tested and only one pollen is responsible for the patient's disease an extract of this pollen only is used in the de-sensitization of the patient. If the patient reacts to several pollens an extract is made containing the pollens in the proportion as indicated by the reactions, and this combination is used in the treatment.

The modern accepted way to treat these patients is by de-sensitization and immunization against the exciting pollens. The rationale of this is based upon the following fundamental biological fact. A protein sensitive animal when injected with a sublethal dose of protein will manifest mild symptoms of shock and if it does not die will not react to subsequent larger doses of the same protein. This refractive period has been called period of desensitization and anti-anaphylaxis. This period is temporary only as the animal again becomes sensitive after an interval of time. This occurs in hay-fever treatments, the patient will experience a brief period of relief from the injections. For this reason the treatments should be scheduled so that a patient is getting injections up to the time of the hay-fever season. If this brief period of relief were the only value of the injections it would hardly seem worth while. A dual effect is obtained from the injections and this is in the nature of a permanent immunization to the pollens, as about 7% of the hay-fever cases are permanently cured with one series of treatments while in the remaining number there is a decreased sensitization to the protein and the patient can receive much larger doses in the treatment the second or third year.

Pollen extracts are prepared in the laboratory for the treatments and diagnosis of the hay-fever cases. The technic used in preparing the pollens is briefly as follows:

In the treatment of cases 1-100 dilution is the weakest solution used. This is done by employing a tuberculin syringe, giving .01— .03— .05— .07 and upwards to 1cc. All injections are given in the same anatomical location, *i. e.*, arm or thigh. I have noted a distinct local tolerance to the extracts from the injections and large doses can be endured without reaction if this rule is followed. If large doses are given in areas not previously desensitized severe reactions may occur. Treatments are given intravenously to those cases that have been under treatments for some time and which have received the maxim dosage of 1cc. of 1-100 dilution subcutaneously.

The first dose intravenously being .02cc. of 1-100 dilution and the dosage increased as indicated by the reactions of the patient. The

intravenous method is not without some reactions in some cases but the results in the cases so treated have been gratifying. This is a preliminary report of the intravenous injection upon a limited number of cases. The intravenous administration of antigens in desensitization treatment should not be generally advocated, and further experimentation is necessary with this method of treaement.

The following results have been obtained in 130 cases of treatment as above indicated:

	Total relief	Some relief	No relief.
First year	7%	63%	30%
Second year	16%	64%	20%
Third year	23.6%	69.4%	7%

Perennial hay-fever Some patients give no history of seasonal periodicity, suffering at irregular intervals thruout the year. It requires real detective ingenuity to determine the cause in these cases. The causes are many some are due to foci of infection particularly of the intestinal tract. Endocrine disfunction is the eitological factor in some cases while others are due to protein sensitization. The proteins responsible for this variety of hay-fever must be sought for in food, bacterial proteins and animal emanations. Sometimes it is necessary to visit the patient's home in search of possible animal emanations as occurs in rugs and furs and pillows. Occupation frequently gives a clue to the etiology. This is illustrated in the case of a man, age 35, who had been suffering from sneezing and itching, conjunctival and asthmatic attacks for years without seasonal recurrences. His occupation was that of a baker, but he had been unable to do his work for a year. The mere passing of a baker-shop would precipitate an attack of hay-fever. With this history food proteins were used in testing him. The patient reacted violently to wheat, rice, barley, rye flour, oats and corn meal. The mere testing the patient by the cutaneous method precipitated a violent attack of hay-fever. In the above case search for the etiology was simple enough but not so in all cases, and not infrequently the cause is never found. Many cases of rhinorrhea are tested at the hospital with sensitization apparently responsible for about 10 per cent of cases.

ASTHMA.

Pulumonary asthmatic attacks are of three distinct types.

First: Anaphylactic spasmodic contraction of the bronchial musculature.

Second. Bacterial infective asthma.

Third: Combination of sensitization and infection.

The first is due to the deposition of the protein upon the respiratory tubes of an animal sensitive to a protein. Spasmodic asthma is true anaphylactic asthma and is a local manifestation of a general condition the same as hay-fever. In fact hay-fever patients will suffer with asthma if the pollens to which they are sensitive reach the bronchial passages. The proteins responsible for this form of asthma are various, animal emanations, food and bacterial proteins, various powders and dusts. The animal emanations most commonly responsible for the spasmodic asthma are from horse, dog, rabbit, chicken and goose. The foods responsible for most of the asthmas of children are wheat, rice, flour, eggs and milk. Bacterial protein sensitization asthma has been rare in our clinic, in fact we seriously doubt if sensitization to bacterial protein exists. This phase of our work has been most unsatisfactory and we are inclined to think bacteria are responsible for asthma as infective agents only. There are a multitude of powders which at times cause asthma and the one responsible is most illusive and difficult to find in some cases. The age of the onset of asthma is very important. The younger it develops the more likely it is a sensitization asthma. If the asthma has developed later in life the more likely it is of the infective type. The treatment of anaphylactic asthma consists of elimination of the offending protein or the de-sensitization and immunization of the patient to the extracts made from the toxic substances. Operations upon the nose and throat will have little value when a patient has asthma of the purely anaphylactic variety.

Second: Infective bronchial asthma. Classically a pyogenic inflammatory infection of the bronchi mucosa, lymphoid tissue and musculature of the bronchi by bacteria. These bacteria and their toxins periodically irritate the vago-constrictor mechanism of the bronchiles producing a spasmodic obstruction of respiration. The infective type also produces a chronic obstruction to the bronchi by inflammatory thickening of the mucosa of the passages. In the infective types and mixed forms, the treatment consists of drugs, non-specific proteins, vaccines, operative procedures, climatic and general supportive measures. Vaccines are of frequent aid in the infective types, while procedures relieving foci of infections in the teeth, sinuses, tonsils and gastro-intestinal tract will entirely relieve some of the cases. Autogenous vaccines are given in strong solutions and in large doses. The vaccines are made fresh every four weeks and treatment continued over a long period of time. A combination of the sensitization and bacterial invasion frequently exists. The sensitization asthmas are prone to become infected. It fre-

quently requires long study and patience to get at the fundamental facts in an asthmatic patient.

ECZEMA.

Some forms of eczema particularly in children have resulted from sensitization to food. The foods responsible are the cereals, eggs and milk. Eczema in adults is also due to sensitization in a limited number of cases. Eczema has been observed in several hay-fever patients. I have had two patients who would have large areas of eczema upon the hands and face during the entire time they suffered with hay-fever. Walker has previously reported the frequency of eczema associated with asthma in children. Immunizaquency of eczema associated with asthma in children. The explanation for the eczematous tendency in sensitive animals has received considerable scientific support. Auer has recently shown that sensitized rabbits will more easily develop an eczematous condition of the ears after application of the skin irritant than nonsensitized rabbits.

URTICARIA.

Urticarial and angioneurotic edema have also been classed as protein sensitizations. I have treated 18 of these cases and have found 7 of them sensitive to some form of protein, chiefly food proteins. Quite a number of my hay-fever cases have developed urticaria during the desensitizing treatments. This is particularly likely to occur if the dose is too rapidly advanced. Experimentally rabbits when injected subcutaneously by a protein to which they are sensitive frequently show urticarial manifestations which may advance to a stage of suppuration and abscess formation. This was first described by [19]Arthus. The differential diagnosis of all sensitizations by means of the precutaneous or intracutaneous tests depends upon the development of an urticarial wheal at sight of the test, this wheal rapidly disappears and the skin becomes normal in from 1-24 hours. Rarely do they suppurate and ulcerate, however it appears that multiple furunculosis is an evidence of the sensitization in some individuals.

CHRONIC CONJUNCTIVITIS.

Chronic conjunctivitis and frequently recurring acute attacks with itching have given sensitization to proteins. I have upon record one case sensitive to salmon and one sensitive to orris root and face powders she was accustomed to using. In both instances the avoidance of these factors have prevented up to this time any recurrance of conjunctival symptoms.

ACUTE GASTRIC COLLAPSE.

My experience with three cases of gastric collapse has convinced me that many patients suffering with acute indigestion of the past

were simply acute manifestations of anaphylaxis. I wish to describe the following case:—A married woman, age 28, in perfect health, became violently ill one morning at 10 a. m. after having eaten ham for breakfast at 8:30. The symptoms manifest were restlessness, vertigo, nauseau, vomiting, diarrhea, acute abdominal pain, cynosis, muscular twitching, rapid respiration, collapse and asthmatic symptoms. At 11 a. m. her blood pressure was 95, pulse 140 and barely perceptible, extreme pallor, dyspnea and exhaustion. Temperature 97.5, leucocyte count 9,000 and .05 per cent polymornuclear leucocytes. Blood count repeated in eight hours, 8,000 leucocytes with neutrophiles 80 per cent. Urinalysis negative. Patient was given adrenalin and strychnine subcutaneously with a rapid respone and recovery. The patient was tested for food sensitization with a marked reaction to pork. She was told to eliminate this element of food and so far has not had any further manifestations of the sensitization. This patient became seriously ill in a brief period of time with symptoms approaching death due to an absorption of an almost lethal dose of protein to which she was highly sensitive. Sensitizations to foods absorbed thru the intestinal tract is somewhat obscure and undoubtedly is due to poor digestive secretions of an excessively permeable intestinal mucosa. Experimental data seems to show that a protein molecule sufficiently hydrolyzed to the peptone or amino stage will not sensitize. Normally the digestive tract should so completely hydrolyze its foods, but in pathological states of the intestines undoubtedly some molecules of the undigested protein is absorbed and acts the same as when artificially introduced intravenously. In children suffering from entrocolitis, [19]Schloss has shown that proteins may be absorbed in an unhydrolyzed state which undoubtedly accounts for more frequent occurences of sensitization diseases in children.

The prognosis of sensitization diseases has been decidedly more favorable since the discovery of their etiology. In every case where there is a probability of a shock factor in the patient, he should have well advised tests made for sensitization. If positive the line of treatment is evident and consists of either elimination and avoidance or an attempt at the active desensitization and immunization to the proteins responsible.

In the treatment of allergic diseases with the etiological protein one must bear uppermost the danger of producing acute shock. The symptoms of shock come on so rapidly and are so alarming to the patient and doctor that they should always be carefully avoided. Several deaths have been reported from the treatment of

these cases. The action of adrenalin in restoring these cases is instantaneous and miraculous in most instances. Treatment of these cases is essentially one of responsibility and the success of treatment and safety of the patient depends upon the care taken. There is a happy medium dosage which is desensitizing and is not alarmingly anaphylactic. I feel, however, that the production of mild symptoms is essential to the successful treatment of these diseases.

CONCLUSION.

There is probably no line of medical research more interesting or more promising than the study of allergy. Practical medicine has again been enriched by fundamental biological studies and simply by the practical application of the fundamental biological truth of anaphylaxis to the etiology and treatment of allied diseases. Diseases whose etiology remained for decades obscure, which offered barren results therapeutically.

CONCLUSION.

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REFLEXES FROM THE RESPIRATORY TRACT.*

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The importance of the respiratory tract in guaranteeing to the lungs proper ventilation, renders it necessary that every part of the body should be rapidly linked up with the lungs and air passages by reflex control. Johannes Müller recognised this when he said "irritation of any mucous membrane of the body is the most frequent cause of reflex manifestations of the respiratory system." Likewise for perfect coordination of the body these air passages and the lungs should be brought reflexly into close accord in the reverse direction with other constituent parts of the body. And it is certainly true that the various portions of the respiratory tract are reflexly related to one another and to the contiguously situated organs, the eye and ear. All reflexes function as a part of the protective machinery of the body. In fact the reflexes which we plan to discuss are those which in the main serve the purpose of guarding the respiratory tract from injury. The appearance of dangerous conditions in the environmental air such as smoke, ammonia, dust, etc., sets up as soon as these substances enter the nasal atrium, reflexes in the nose itself, in the eyes, in the pharynx and larynx whose purpose it is to remove the noxious matter. Also the development of abnormalities of growth or bacterial infection acts as an irritant and sets up protective reflexes.

Reflexes from the Nose. It will perhaps be advantageous for the sake of proper understanding if we quickly sketch the possible type of reflexes which may be involved in the further discussion. The neural structure which underlies the axon reflex represents the most primitive form of nerve connection. Reflex action over such a pathway is possible without a central nervous system. Two forms of neural architecture for axon reflexes have been described, the first in which only one neuron is involved and the second where two neurons synapse in an autonomic ganglion. The first and simplest form is that in which the afferent axon at the periphery gives off collaterals to the skin as well as to blood vessels in the same cutaneous area. A neural structure of this form is the basis for the hyperaemia which occurs when mustard oil is placed on the conjunctiva. This is not a direct action of the irritant since cocaine

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inhibits it. The reaction is therefore brought about by nerve pathways; however, the central nervous system plays no part since section of the spinal roots supplying the conjunctival area, does not preclude the reaction. It has been shown that stimulation of the peripheral end of a severed posterior root will bring about a vaso-dilation of the area enervated by that root. This does not prove that posterior roots carry efferent fibres but as Bayliss supposes, the neurons involved in the reaction are of the ordinary afferent type capable of conveying not only sensory impulses into the cord from the blood vessels but also able to bring about vaso-dilation as well. This would seem to indicate that if impulses, sensory in character, originating at the surface of the body can spread throughout the neuron and all its collaterals, and if one of these collaterals were in relation with the blood vessel of that area, then stimulation of the surface would bring about vaso-motor changes without the intervention of the central nervous system. The reaction is one mediated within a single neurone and is therefore called an axon reflex, one collateral acting afferently and another efferently.

The second type of axon reflex involves a post ganglionic neuron passing through an autonomic ganglion and giving off, on its way to the periphery, a collateral which synapses within the ganglion with another postganglionic cell body. Stimulation of the former neuron at the surface of the body would, through its collateral synapse discharge the second postganglionic neuron and thereby initiate a peripheral effect.

Reflexes involving the central nervous system as centers are either purely visceral, i. e., autonomic, or mixed somatic and visceral. In the former type the afferent neuron is a sensory visceral cell such as the taste fibres and the efferent neuron an autonomic, as for example the secretory fibres to the submaxillary gland. The neural architecture of the mixed reflex would include an afferent visceral pathway like that from the nose to the pons and an efferent somatic fibre conducting from the center to the respiratory muscles, the reflex being involved being a sneeze or cough. This latter is the usual sensori-motor reflex reaction involving striated muscle.

Anatomy of the nasal reflexes. The nasal mucosa receives its visceral afferent supply from the trigeminal sensory divisions, the ophthamic and maxillary, principally the latter. These fibres would constitute the afferent arc in reflexes from the nasal cavity. Impulses originating in the septal mucosa as the result of some irritation in this region would pass via the nasopalatine nerve and through

the sphenopalatine ganglion and then to the maxillary division of the trigeminal by means of the sphenopalatine nerves. Following the maxillary nerve to the trigeminal ganglion (the gasserian) the impulses enter the pons by means of the sensory root of the trigeminal nerve. From other portions of the septum impulses would be conducted via the internal division of the nasociliary branch of the ophthamic nerve to the trigeminal ganglion and from thence to the pons as before. From the lateral wall impulses traveling over the nasal and palatine nerves reach the sphenopalatine ganglion. The pathway from this point would correspond to that outlined for the nerves of the septum. It must be remembered that the sphenopalatine ganglion belongs to the autonomic system. Passing through it are the afferent fibres just mentioned together with efferent autonomic neurons, cranial and sympathetic. The cranial preganglionic fibres, originating in the motor nucleus of the seventh nerve in the pons, leave the nerve as it passes through the petrous portion of the temporal bone as the great superficial petrosal which joins the vidian nerve to reach the sphenopalatine ganglion. These preganglionic fibres synapse here with post ganglionic cell bodies the axons of which are carried in the palatine nerves to supply the blood vessels and mucous glands of the nasal mucous lining. The sympathetic (cervico-thoracic autonomic) postganglionic neurons arise in the superior cervical ganglion. They pass to the internal carotid plexus and from thence travel via the great deep petrosal nerve and vidian nerve to the sphenopalatine ganglion; from this they are distributed through the palatine nerves. It is probable that the post ganglionic fibres from the sympathetic autonomics in passing to the septal mucosa through the sphenopalatine ganglion give off collaterals which synapse with the cells of origin of the postganglionic fibres of the cerebral automomics. This gives us the neuron architecture of an axon reflex of the second type.

Central connections. We have just seen that the trigeminal nerve carries afferent conducting fibres which pass into the pons. Here they divide into an ascending branch terminating in the main sensory nucleus; the descending collateral runs through the spinal tract and ends in the nucleus of the spinal tract of the trigeminal nerve. This group of cells receiving some fibres from the glossopharyngeal and vagus nerves becomes continuous in the spinal cord with the substantia gelatinosa Rolandi. There is consequently a continuous column of grey matter extending from the sacral region in the cord to the pons and capable of acting as a receiving station from the afferent fibres of the trigeminal and a discharging center to prac-

tically all segments of the cord. From the cells of the main sensory and spinal nuclei of the trigeminal nerve, fibres arise which enter the reticular formation and are grouped into longitudinal bundles from which collaterals are given off to the motor nuclei of the brain stem, thus establishing reflex connections with the efferent fibres of the cranial nerves. Ascending fibres enter the main sensory tract of the opposite side which ends in the thalamus, and synapse with sensory radiations to the cortex. By this means sensation becomes a component part of certain reflexes such as the sneeze and cough.

It thus becomes apparent that with the possibility of such wide spread interneural connection in the pons between the sensory afferent somatic and visceral fibres on the one hand and the motor nuclei of origin of all the cranial nerves carrying motor fibres (somatic and visceral) as well as those in the cord via the substantia gelatinosa on the other, reflexes to practically all parts of the body may be initiated from the nose.

General functions of the nose. We must pause just a moment to consider the rational and the function of various parts of the respiratory passages. The nose functions as the atrium of the air passages. Its olfactory epithelium was developed primarily as the receptor surface or sensifacient area of a protective reflex by means of which we might with the aid of the olfactory sense tract and cortical center, become conscious of the proximity of dangerous gasses or air particles. The nasal mucosa not concerned with olfaction has for its purpose the secretion of fluid which raises the water vapor tension of the air inhaled until it becomes practically saturated. The massive blood supply to the mucosa allows of ample heat radiation by means of which the temperature of the inspired air becomes that of the body. The hairs or vibrissae of the vestibule act as filters for large particles of dust and bacteria carried in from the inspired air. The ciliated epithelium completes the task of removal of the bacteria by assisting the fluid secreted by the eyes and nose to flush the surfaces of the cavity. It has been said that the nasal mucous lining may show bacteriocidal properties. Air reaching the naso pharynx should be practically sterile. In mouth breathers removal of bacteria occurs in part by the moist surfaces of the mouth and pharynx. Since the nose is normally the main point of entry of gaseous substances to the body, it is quite understandable that it should be reflexly coordinated with all parts of the respiratory tract and of the body as a whole.

It may be interesting in connection with the thought of the protective function of the nose to recollect the reaction which occurs

as an acute rhinitis, a prodromal symptom in so many infectious diseases, i. e., influenza, measles, whooping cough, scarlet fever, diphtheria and erysipelas. The gastro-intestinal tract reflexly attempts to rid or purge itself of poisonous substances by vomiting and diarrhoea. Apparently in an analogous way following an infection which usually enters via the respiratory tract the body sets up reflex reactions which would serve to remove the source of infection if it happened to be nasal in character. The vaso-motor dilation succeeded by the perfuse discharge which is at first watery then mucous and only later becomes mucopurulent, must be considered as a protective or flushing mechanism. That this coryza-producing function of the nose is controlled by reflexes from the surface of the body has been shown experimentally. It is possible to alter the blood supply to the nasal mucosa and thereby the temperature of the nasal cavity by changing the environmental temperature affecting the cutaneous surface of the body. If the temperature of the outside air be lowered, the heating function of the nose is increased by a vaso-dilation of the nasal mucosa. This increase in blood supply is sufficient to materially augment the thickness of the nasal mucosa and to produce a profuse mucous secretion. Even normally the hyperaemia may be sufficient to obliterate the lumen between the turbinate and the septum. This bringing together of two surfaces acts as a stimulus sufficient to originate a reflex such as a sneeze so common in these conditions.

Intranasal reflexes. Francois Franck in 1889 stimulated with an electric current the edge of the middle and inferior turbinates and obtained reflex hydrorrhoea, thus proving the possibility of bringing about intranasal vaso-motor reflexes from irritation of the mucosa. The axon reflex finds its exemplification in the hyperaemia and rhinitis which are present and quite common amongst furriers, millers, workers in saw mills and with tobacco, iodine and bromine. The irritants present in the air of places where these trades are conducted, initiate the reflex vaso-motor response.

Mechanical pressure might be mentioned as one other cause of vaso-motor changes from the nasal mucosa. The turbinates normally are in rather close proximity to the septum. Not more than 1/8 to 1/4 of an inch of air space intervenes. The air passing through this channel takes up moisture. The drying in itself acts as a stimulus, but the narrower the interspace as in deflected septa, bony and cartilageous outgrowths, thickening of the mucosa of the turbinate or septum, and polypous growths from the turbinates, the more rapidly the air moves; in fact, in some instances sufficient

contact to cause irritation may be established betwen septum and turbinate. This stimulates the sensory nerve endings in the mucosa and produces reflex effects the more prominent of which are sneezing and coughing. These abnormalities may not in themselves originate the reflexes but only in virtue of the hyperaemia which they produce. Persistent sneezing with watery discharge is held to be symptomatic of disturbed septal-turbinal relation.

Reflexes initiated from the nose with effector surface elsewhere in the body. Respiration is markedly affected by nasal conditions. Cold water on the skin of the nose as well as sudden chilling of the nasal mucosa inhibits respiration completely and in whatever state of the respiratory cycle the stimulus may be applied. Diving animals show this reflex to best advantage. Strong irritation such as ammonia fumes likewise causes stoppage of breathing, while the electric current markedly augments the rate and depth of respiration. Tickling of the mucosa or the pulling out of nasal hairs will cause reflex sneezing. These are protective reactions to prohibit the entrance of injurious substances into the respiratory tract. Hazelhurst has described a case with frequent attacks of sneezing always associated with mastication and ingestion of foods. The symptoms disappeared following removal of a septal ledge which pressed against the inferior turbinate actually indenting it. John Mackenzie mentions certain sensitive areas from which it is particularly easy to elicit a reflex response. These areas are the mucous membrane covering the middle and inferior turbinates and posterior half of the septum. Jacobsen's tubercle lies at the anterior end of the middle turbinate. It will be noticed that these areas embrace those portions of the nasal surface devoted to turbinates which lie in intimate relation with the septum and in which we find the large venous plexuses. This large blood supply would account in part for the normal heightened irritability of these areas.

That the hyperaemia is an important feature in the origin of the reflexes seems to be proven from the fact that in atrophic rhinitis, reflexes are not prevalent, although the accumulation of crust may become an exciting cause.

Many observers speak of reflex cough from nasal abnormalities such as polyps, hypertrophic rhinitis, septal spurs and deflections and irritations of the anterior and posterior extremities of the inferior turbinates and inner surfaces of the middle turbinates, the so-called "cough-spots" of Francis. It is evident that these conditions are similar to those that produce hyperaemia so that the cough which results in some cases is merely an extension of the reflex arc to an area extra nasal. It is not possible to give a reason why

these pathological conditions do not always act as excitants to the cough reflex. One might imagine a hyperirritable center as in some neurotic condition, or some abnormal condition of the larynx, or both nose and larynx together might serve as points of somewhat low threshold, neither of which alone capable of eliciting the reflex cough but both functioning synergetically, able to discharge it.

The possibility of nasal asthma must not be overlooked. The reflex relationship between the nose and bronchial musculature was emphasized by Lazarus who repeating Francois Franck's work, stimulated the nasal mucosa with electrical currents and obtained contraction of the muscular coats of the bronchioles as evidenced by increased intrabronchial pressure. Asthmo-genic points, according to Brugelmann, are small hyperaesthetic areas in the region of the middle and inferior turbinates, of different color than the rest of the mucosa. Brodie and Dixon caused a spasm of the bronchial musculature by stimuli applied to the septum. When patients give a history of attacks of sneezing, Lorie advises that the nose be examined as a possible seat of the asthma. Cures of asthma are reported by many authors when nasal abnormalities are corrected. The conclusion to be drawn from these statements should be that in asthma refractory to treatment based upon anaphylaxis, the nose might well be explored.

Mention in the literature has been made of cases of excessive salivation cured by nasal treatment. Whether the salivation was the manifestation of some unrecognized central lesion or whether the reflex was direct, it is difficult to decide. Anatomically it is possible. The same train of critical thought must accompany the following statements of like nature. Richet reports tic doloreau cured by removal of a growth from the septum. Sallinger has seen cases of epilepsy apparently due to intranasal disease. Optic abnormalities are deemed to be caused reflexly by nasal conditions together with dysmenorrhea, photophobia, conjunctivitis, asthenopia, epiphora and glaucoma. Many of us may rightly be somewhat skeptical concerning the direct reflex relation between the nasal cause and the syndrome effects just mentioned. It is not my purpose to present briefs in favor or against, but merely to state that anatomically and functionally the reflex effects are possible.

It is always well to be mindful of the fact that in hypersensitive conditions of the central nervous system, disturbances of reflex origin may manifest themselves from small and, under normal circumstances, insignificant causes. The wide spread central connection in the pons of those fibres from the nose through the trigeminal nerve apparently allows relatively slight alterations in the mucosa to

show greatly varying effects according to the state of irritability of the central nervous system. Thus for example, sneezing, coughing, glotic and broncheal spasm are more common clinically in neurotic individuals. In fact Bosworth stated that extreme tendencies to intranasal congestion when the hyperaemia became pronounced, was indicative of a neurotic temperament.

Finally the subject of nasal misreferred pains must not be overlooked even though they are not strictly reflex phenomena. The nasal mucous membrane is not a place of high pain sensibility. According to Head, impulses originating on such a surface and capable of bringing about painful reaction in the cortex would be misreferred to the cutaneous surface as a place of higher sensibility. However, on account of the great area of distribution of afferent fibres in the 5th, 7th, 9th and 10th nerves and their close central connection, any low threshold point will bring about a misreference over a wide spread cutaneous area. The pains of trifacial neuralgia in atrophic rhinitis are examples of this, as well as the extensiveness of pains arising from a single tooth. Sluder has spoken of a sphenopalatine syndrome of irritation, pain behind the eye and mastoid which radiates down into the neck and arms to the fingers. It is stated by Thomas that sinus disease causes a painful area referred to the ear, while in simple acute rhinitis the pressure exerted on the sinuses is responsible for the frontal headache through misreference. In certain cases of high deflection of the septum, headaches may appear from negative pressure in the sinuses developed as the result of absorption of air from the closed spaces.

Reflexes from mouth and pharynx. The teeth are supplied by afferent sensory fibres from the dental branch of the trigeminal; the mouth and pharynx, from the pharyngeal plexus, palatine branches of the sphenopalatine, glossopharyngeal, vagus and the sympathetic autonomics. The intimate central connection of these nerves has been previously mentioned. It may be advisable however, to emphasize the fact that the afferent glossopharyngeal fibres entering the medulla along the ventral side of the restiform body help to form the longitudinal tract called the fasciculus solitarius; the lower part of the nucleus of the tractus solitarius is the respiratory center. These central connections are sufficiently complicated to allow of the possibilities of reflex pathways from the mouth and pharynx to all parts of the body.

The teeth may act as receptor surfaces for reflexes appearing over widely divergent areas and with greatly dissimilar symptoms. Kirk states that dentitional reflexes may include derangements of

speech, hearing or vision. He mentions cases of strabismus among school children as well as chorea, epilepsy, dementia precox and intellectual dullness as the results of improper care of the teeth, and finds that these manifestations disappear after dental treatment. Clark gives as causes of increased dental reflexes, infection of gums, decay of teeth, impaction, faulty dental work. From these irritations come reflex neuritis, neuralgia, insomnia and mental disorders.

Concerning the wide area for misreference of pain sensation in the case of the teeth we are all familiar. This requires no further discussion save merely to suggest that while the teeth may be the originating area for the pain, all facial pains should not be regarded as sufficient evidence for advising teeth removal.

From the pharynx there is elicited the respiratory or breath stopping reflex which occurs when food is on the rear of the tongue preparatory to swallowing. The stoppage of the respiration takes place in any phase of inspiration or expiration. Stimulation of the central cut end of the glossopharyngeal nerve will cause inhibition of the respiration in the passive phase of expiration. The reaction is of course protective and allows the various muscular contractions to supervene in order that the Eustachian and nasal orifices as well as the glottis may be closed so that the food may be shot through the pharynx. The pharyngeal mucosa is particularly responsive to light pressure and stroking, and reflex vomiting or gagging may easily be initiated by placing the finger in the pharynx. The sensitiveness of the mucosa to such treatment varies with different individuals and its absence is well known to be a symptom of tabes. Irritation of another type elicits from the pharynx reflex cough, or hacking. The pharyngeal tonsils especially may function as receptor surfaces for reflexes when they are inflamed through the presence of bacteria or pus. The embedded form is apt to result in more reflex effects because they cannot enlarge greatly without causing local disturbances which act to initiate the reflex. Enlarged lingual tonsils may similarly function as an excitant while pharyngeal reflex cough may also originate as the result of an excess of mucous material from the post nasal pharynx flowing over normal or somewhat inflamed and irritable tonsils. Mackenzie suggests an excessively anterior scrolled epiglottis as a provocative agent of the cough.

Reflexes from the larynx. The larynx is supplied by the superior and inferior laryngeal branches of the vagus nerve but the former nerve carries all the sensory or afferent fibres whose cell bodies

are in the ganglion nodosum; the central axons entering the pons descend and joining afferent fibres from the glossopharyngeal, form the tractus solitarius and terminate in its nucleus the lower part of which, according to Cajal, is identical with the respiratory center. From the nucleus of the tractus solitarius, descending fibres arise, the tractus solitarius spinalis, which pass in the cord to the primary motor neurons belonging to the phrenic and intercostal muscles. It is thus possible to trace the nervous pathway over which impulses must pass in the production of the reflex cough from the larynx.

Stimulation of the central cut end of the superior laryngeal nerve, will cause the respiration to stop in the phase of passive expiration. If the stimulation is sufficient the bronchioles may also become markedly constricted and cardiac reactions may also appear. This protective response of inhibited respiration can be originated as we have seen from the nose, pharynx and larynx.

In this connection it may be worth while to comment upon the possibilities inherent in the injury under surgical procedures of the superior laryngeal nerve. All are familiar with the dangers especially present when for some reason or other not frequently recognized, a state of hyper-irritability of the larynx exists. This may account for some of the disasters of bronchoscopy although it cannot bear the burden of all the fatal results. There are many cases which do not, unfortunately, get into the literature, of death occurring after some operative treatment in the neck especially around the thyroid gland or larynx. Two symptoms, usually not appearing in the same case, are prognostic of laryngeal afferent injury. In one, the respiration stops for no evident reason and quite suddenly, with the heart apparently in good condition. Attempts at artificial respiration are unsuccessful, a few gasps may result, the heart during this time remaining fairly normal. Gradually however, in spite of all attempts to start respiration, the heart becomes weaker and death occurs with some asphyxial spasms. Quite evidently a reflex inhibition of the respiratory center has occurred, sufficient in degree to repel all attempts to discharge it. In other cases, the heart begins to slow down; breathing may apparently be unchanged. The pulse rate may continue to decrease or the heart may stop suddenly before any respiration disturbances would seem to warrant such cardiac changes. Here of course, there is no evidence of asphyxial conditions. It would seem that the laryngeal trauma has brought about reflex inhibition of the heart.

Coughing which also may be elicited from a large part of the respiratory passages, must be considered as a remedial as well as

a protective process. For diagnosis it possesses the same value as pain, and should be employed similarly. But just as pain has a different protective and remedial value, so has coughing. Hence the use of antibechics is contraindicated in many instances. Chevalier Jackson calls the cough the "watch dog of the lungs" and he reasons that this sentient protector should not be thoughtlessly put to sleep by narcotics. He also asks whether pneumonia may not follow influenza as the result of the inability on the part of the lungs to rid themselves of some of the toxic material through depression of the cough reflex. Also he suggests that post operative pneumonias may possibly be caused by the temporary abolition of the cough when a general depressant like morphine is employed.

It is questionable whether the trachea and smaller bronchioles may participate in a cough reflex. Nothnagel mentions that irritations of the laryngeal mucosa above the vocal cords produces spasmodic contraction or closure of the glottis but no cough. But coughing does occur through irritation below the cords. The most sensitive area is the interarytenoid mucous membrane and the bifurcation of the trachea, (the carina). Sensitiveness to the cough reflex decreases from the larynx down to the pulmonary parenchyma. There is a teleological reason for this since the further down the trachea the foreign substance resides the less effective is the action of the cough. Mackenzie agrees that only when the mucous reaches the upper trachea or larynx does the expulsion take place. He cites the absence of cough in pneumonia as evidence of this. Many will disagree with this statement and other explanations must occur to every one. Amongst bronchoscopists the opinion is held that at the carina or ridge at the bifurcation of the trachea resides a spot extremely sensitive to irritation and acting as a receptor surface for the cough reflex. Some years ago, Imperatori carried on some work with the idea of attempting to explain the cause of sudden death in certain cases where bronchoscopic examination was in evidence. The results of this investigation were presented to this Society in 1916 as a candidate's thesis. Imperatori showed that stimulation of the carina caused increased rate and amplitude of respiration. From other findings the conclusions were tentatively advanced that sudden pulmonary collapse might reflexly inhibit not only respiration but the heart as well. The question still remains unanswered and an enticing field of work still awaits an enthusiastic laryngologist.

Laryngeal spasms of reflex origin. That spasms of the adductor muscles originate as the result of reflex stimulation, seems to be

well established. In the young individual it may occur from a great variety of causes all acting to elicit the reflex, but in the main the neurotic factor is to be emphasized. In pertussis the adductor contraction is said to be the result of central stimulation; but in most of the other conditions the sensory area from which the reflex originates is the larynx, nose or pharynx. It appears to be true that local disturbances in other parts of the body when occurring in connection with a neurotic condition may also act as casual excitants. In adults the so-called nervous laryngeal cough is to be explained in a similar way. It is interesting to note that this reflex generally ceases at night or when the patient's attention is diverted. This introduces a matter of some importance concerning the mechanism of the reflex involved in the cough. Jendrassik divides all reflexes into the spinal type which includes the tendon, periosteal and joint reflexes, the cerebral types such as the cutaneous reflexes and the complex type of which sneezing, coughing and vomiting are examples. One characteristic of the latter two types as distinguished from the tendon reflex is the necessity of sensation of tickling in connection with eliciting the reflex. Apparently sensation, or so-called cerebral component is necessary to the completion of the reflex. This raises the question as to whether the cough may be aroused without consciousness. Sahli, in his diagnostic methods, states definitely that "completely unconscious individuals never cough or sneeze." This raises the further question as to what is meant by complete unconsciousness. Some observers speak of the degree of unconsciousness or anesthesia necessary to inhibit the cough. It is to be assumed that in the last stage of anesthesia, reflex coughing could not occur since the muscles are thrown out of action; but in the opinion of anesthetists the individual may cough in light surgical anesthesia. Imperatori, at my suggestion, recently examined eighteen cases in anesthesia with the view of determining the relation of the disappearance of the corneal reffex to the ability of eliciting the cough. He informs me that coughing may be reflexly produced after the cornea has lost its reflex. In this he agrees with Wright and Smith who also state that the posterior wall of the larynx is most susceptible to irritation. Whether the pharynx or larynx first ceases to serve as a sensifacient area for the cough and how deep must be the anesthesia in order that the reflex drops out, Imperatori is unable to decide at present. If all the above is true the statement of Sahli must be incorrect. It had always been my assumption that coughing during supposed sleep was the result of

the irritation which brought the individual into a state of semi-consciousness during which the sensation of tickling actually came into consciousness and the cough resulted. The absence of the nervous laryngeal cough in individuals when their attention is diverted seems to add some evidence to the view that sensation is a component in production of the cough, though it must be admitted that the subject is still an open one.

Another condition of laryngeal origin is the vertigo which sometimes accompanies the abductor spasm of the glottis. Among other indefinite etiological factors are mentioned tobacco and alcohol. Apparently the vertigo is merely a symptom induced by impaired cerebral circulation resulting from interference with blood flow in the lungs from the cough.

In certain cases of acute laryngeal oedema the disturbance may also be considered in the same light as the production of a mucous oedema of the nasal mucous membrane, either as the result of a pure reflex involving central synaptic connections in the medulla or through an axon reflex as explained before.

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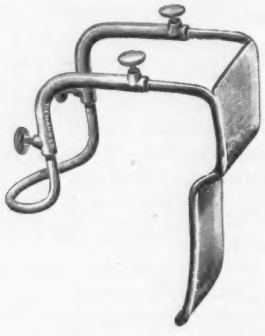
A SELF-RETAINING TONGUE-DEPRESSOR.

Dr. Arthur J. Herzig, New York City.

During the year 1904 Tiemann & Co. constructed an instrument for me to be used as a self retaining tongue-depressor. This instrument consisted of 4 parts. One, the upper, to be placed upon the dorsum of the tongue; the lower to be placed under the chin. The two parts were joined together with two L-shaped parts and held in place by 4 screws. All the screws were to be tightened when the instrument was in place. The present instrument is a replica of the old one with the exception that I have had a solid plate placed upon the tongue part (upper) so that the tongue would not raise through the fenestrum. The report of the first instrument was published in the *N. Y. Medical Journal,* April 6, 1907. I have used this instrument almost continuously for the past 18 years and have found it of the greatest assistance in removing tonsils under local anesthesia, also under general anesthesia. When a view of the lower part of the tonsil is required, why the instrument which is already in place is tilted backward and a view of the lower tonsil is obtained, also the tip of the epiglottis. It may also be used in any throat operation where the immobility of the tongue is desired. By reversing the parts it may be used as a simple tongue depressor. Many thanks are due to Tieman & Co. who were kind enough to assist me in the mechanical details of this instrument.



229 West 78th Street.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

A stated meeting of the Philadelphia Laryngological Society was held Tuesday evening, May 2, 1922, at 8:15 P. M., Cadwalader Hall, College of Physicians.

Three Cases of Mastoid Conditions. Dr. Henry Dintenfass.

The three cases I wish to show the Society are anything but rare, but as they represent distinct types of mastoid conditions, are of sufficient importance to merit our thought and attention.

The first case presents a bilateral mastoid with a complication before operation. The second is interesting on account of the complication following the operation. The third shows an extensive mastoid involvement following a tonsil and adenoidectomy.

Case 1. Evelyn W., age 2½ years. After a severe naso-pharyngeal infection developed a double acute otitis-media with a profuse purulent discharge. She ran a varying temperature from 99° to 102° for several weeks, during which time there was no mastoid tenderness and no sagging of the posterior wall on either side. After this period her symptoms subsided. Temperature remained normal, discharge began to get less, appetite got better and the prognosis for a complete cure seemed quite favorable. For two weeks the apparent improvement continued, when suddenly one morning the mother noticed a slight drawing up of the corner of the mouth when the child laughed. This condition grew rapidly worse; and, when I saw the patient several hours later, a full blown facial paralysis was present. This became still more complete by the next day, the day of operation.

Removal of the cortex on the paralyzed side disclosed considerable necrosis of the mastoid process, with some exposure of the middle fossa. We decided then to open up the other side and were more than surprised to find almost the same pathology present.

The facial paralysis began to improve on the 6th day following operation, complete recovery taking lace in four weeks.

The literature of this type of facial paralysis is quite meagre. Most men believe that the deshiscence in the bony wall of the canal just at the point of the bend between the horizontal semi-circular canal and oval window, permit the presence of the exudate to produce pressure on the nerve. This is particularly true in children and infants. However, the theory might be easily advanced that even in those cases where the canal is intact, the necrosis of the mastoid by extension produces an ostitis of the canal proper which in turn presses upon the nerve sheath, causing paralysis.

The lesson to be learned from this case was that there was no positive indication for operation, until the facial paralysis made its appearance. Its occurrence always means immediate surgical intervention.

Case No. 2. Lillian C., age 14 months. History of influenza followed by a right-sided acute otitis media with spontaneous rupture of the drumhead. Three weeks later swelling and external fluctuation back of the ear.

I operated on the baby on November 7, 1921. The involvement was moderately extensive. The lateral sinus was exposed. Examination of the pus showed mixed streptococcus. The patient was returned to her room in good condition, and for two days the temperature was normal. On the afternoon of the third day following a chill the temperature rose to 104°. The fourth day it came down to 99° in the morning, while in the evening it rose to 104½°. On the fifth day the temperature went up again after another chill to almost 105°. At this point the blood count showed 37,000 leucocytes. The differential count being, small lymphocytes 24 per cent, large lymphocytes 2 per cent, polymorphonoclear 73 per cent. There was nausea and vomiting. Examination of the wound showed that it was in fairly clean condition with the exception of a

point just over the lateral sinus, where a slight patchy exudate was present. A blood culture was taken and proved unsatisfactory. Naturally the thing that suggested itself to me, and was uppermost in my mind, was a lateral sinus complication. There was apparently no tenderness along the course of the jugular, and so we thought we would procrastinate for twenty-four hours before we decided to do anything really radical. On the morning of the sixth day following the operation, much to my relief I discovered a suspicious redness and bogginess of the lobe of the ear, which looked like erysipelas, and which proved itself to be such. The temperature now, while still remaining high, assumed a more even character.

The wound was dressed with weak bichloride dressing, which also covered the erysipelatous area, and after a week the patient progressed to an uninterrupted recovery.

On looking up the records I found that erysipelas of the mastoid is more common than after any other surgical operation because of the similarity of the etiology of both infections, the streptococcus.

According to Phillips, chills and extremely high temperature which for 48 to 72 hours precede the cutaneous flush, are most puzzling and are always suggestive of intracranial complications. To my mind, the infection gains entrance through the skin sutures. For that reason, in infants and young children, I have followed the procedure of using no skin sutures whatever, leaving the wound entirely open.

The lesson from this type of case of course is evident. Had the lateral sinus been opened the chances are the case would have gone to a fatal termination. It shows the need of proper study and careful watching before anything radical is decided upon.

Much has been written in the last several years with reference to pulmonary abscess following removal of tonsils and adenoids; one scarcely picks up a periodical on oto-laryngology without some mention being made of the subject. Nothing, however, or very little, has been said on the question of mastoid abscess after tonsil and adenoid operation. For that reason and because there seems to be some similarity in the causative factors between the two I will briefly report—

Case No. 3. Edward L., age 20, was advised by me to have his tonsils and adenoids removed for a possible improvement of a mixed deafness, a condition which he stated had existed since childhood. The tonsils were moderately enlarged, but showed nothing unusual. However, post nasal examination of the naso-pharynx disclosed the adenoids to consist of an immense mass of lymphoid tissue which completely filled the vault, and bled quite easily when palpated.

I operated October 1, 1921. The removal of the tonsils was accomplished by the La Force instrument, little or no bleeding resulting from them at the time or subsequent to it. The adenoid extirpation was done with La Force adenotome, after a thorough separation of the mass from the pharynx with the index finger. Considerable hemorrhage occurred, which was controlled fairly easily. Two hours after the operation, the adenoid hemorrhage started afresh, making its appearance as nose-bleed, which became quite aggravated by patient repeatedly blowing his nose. However, the bleeding was stopped without the necessity of a post-nasal tampon being used and the patient left the hospital the next day in good condition.

On the third day after the operation earache with stuffiness and tinnitus was complained of. Examination post-nasally showed both eustachian eminences intact. The pain steadily increased, until several days later, when spontaneous rupture of the drumhead occurred, he absolutely refusing to have a paracentesis performed. Mastoid swelling and tenderness developed and symptoms gradually grew worse until I was forced to do a mastoid operation three weeks later.

The points I wish to emphasize here are: first, that a mastoid abscess, like a pulmonary abscess, may and does occasionally follow the removal of tonsils and adenoids, despite the fact that we are in the habit of overlooking it; and, second, that the cause of such a complication, while it is generally believed as being due to infection traveling up the eustach-

ian tube, the great majority of cases have given histories the outstanding feature of which is, as in this case, of excessive adenoid bleeding. The blood from the nasopharynx forces its way into the tympanic cavity through eustachian tube, and having gained entrance acts like a foreign body. A bacterial invasion evoking a purulent exudate does the rest.

DISCUSSION.

DR. HARRY A. SCHATZ: I have in mind a little baby where facial palsy developed early as a complication and I asked my chief in that hospital what chances there were if the condition were operated on. I was told that in acute cases like that, possibly it would not do any good. I had my mind all set that I would be told that early operation would be the thing. I would like to know what is right, the conservative or the radical.

DR. — — MACFARLAND: Two years ago, I operated on a patient about eight or nine years old who had a chronic mastoid following scarlet fever when he was an infant and he had a running ear ever since. When he was brought into the hospital he had a facial palsy. I advised operation immediately and did a radical mastoid. The entire bone canal of the facial nerve had been necrosed and had separated from the other half of the canal so that we had in one half a sequestrum and the other good bone. The sequestrum was removed about three-quarters of an inch along the canal, for the nerve could be seen very easily in both bones. I left the rest of the bone intact. I gave a rather unfavorable prognosis.

DR. HENRY DINTENFASS (in closing): My opinion would be that operation should be done in these cases to relieve at least part of the paralysis. A very narrow canal when swollen would cause enough pressure to cause paralysis.

"Otologic Aspects of a Case of Myxedema. Results of Thyroid Medication." Dr. Harry A. Schatz.

The reduction in the secretion of the thyroid gland produces a fair number of patients in the nose, throat and ear department, but they are not usually given the consideration from the hyperthyroid state. The diagnosis can be made by just one glance at the patient. This case is where the hyperthyroidism was only a partial one. The patient was a typical myxedema patient who reported to the Ear Dispensary. She had not been treated anywhere else. She was in a very bad mental state; she told me confidentially that she had contemplated suicide. She complained of a peculiar feeling in the head, describing it as a fuzzy-wuzzy feeling in the head and a ringing and buzzing in the ears. Her hearing was diminished. The diagnosis can really be made by inspection. The color is a peculiar yellowish, waxy cold skin, like a mild jaundice color, the lower eyelids are all puffed up and the skin is dry. The tongue is thick and bulky. Even this patient's tongue is not yet normal, although there has been a reduction of the tongue since she has been under treatment. A good many patients who come into the dispensary that have myxedema will show the same peculiar condition of the tongue, but most of them are suffering from a hyperthyroid condition. This patient was first put down as a case of nerve deafness. I recognized her as a hyperthyroid case and put her on treatment. The hearing originally entered in book made by our dispensary assistant was that she heard the fork 4" right, 5" left, on second visit 7" in right and 8" in left. In November, 1921, I started her on thyroid extract. When I tested her on the 25th of April, nearly five months later, her hearing on right side was 11", while the left ear was 10". There was no change in her hearing of the very low tones. Hearing to the Galton whistle was practically unchanged. The biggest change was that hearing for the main fork was double. Subjective symptoms have all vanished, apparently from thyroid extract. When I examined her today (of course the hearing changes a little) it was 8" for right, 10" for left. She has been getting no treatment. I would like her to come in and explain her condition herself. I started her on thyroid extract 1 gr. tablets 3 times a day and used her weight as an index. When she had reduced six or seven pounds in weight below her usual, then I cut her tablets down. When weight remained stationary, I kept at that dose. When she complained of weak-

ness, I stopped the thyroid and gave her strychnin, 3 1 gr. tablets. Everything depended on symptoms and weight. She has had no local treatment for the past five months. She will describe the symptoms she felt referable to the head before treatment was started. Her hearing is now very much improved, the ringing is almost entirely gone and the feeling in the head is all gone.

DISCUSSION.

DR. WM. G. SHEMELEY: I enjoyed hearing this case very much, especially so from the fact that recently I abstracted an article by Dr. Selfridge of San Francisco, in which he called attention to the fact of the endocrines and the poor results that are obtained by certain cases. Dr. Selfridge especially brought out the fact of the poor results following work on the nose and throat and he cites some very interesting cases, one of which was a boy, thirteen years old, who had, I believe, been operated on for adenoids, which recurred several times afterwards. Each time it recurred apparently larger than the preceding time. He then studied the case from the endocrine standpoint and found that there was insufficiency. He then put the patient on thyroid extract and child improved. Investigation still further found some other disturbance and treatment was combined and he was very much surprised to find no recurrence of adenoid. The child had better health and was much advanced in school. There is one question I would like to ask Dr. Schatz. He speaks about his functional hearing test. I heard nothing about his Weber, and Schwabach. Were these tests made?

DR. HARRY A. SCHATZ: My patient was diagnosed as a nerve deafness. That was due to the fact that her air conduction was better than her bone conduction. Bone conduction was below normal; in other words, there was evidence of a nerve impairment.

DR. — — MACFARLAND: One has to be careful about giving thyroid. I saw two cases in which doses of one gr. had been given and apparently nothing happened for a while. I had one case in which I started with one gr. and kept it up for some time. This case also went bad with these symptoms. They had severe palpitation, coming on suddenly, apparently some edema of the throat with a suffocation and altogether alarming symptoms. Fortunately, they cleared up rather promptly in a day or two. The tablets are put up in ¼ or ½ gr. doses. There seems to be a tendency of accumulation of that dose even though you are running along and not increasing it.

DR. R. F. RIDPATH: My experience in myxedema is practically nil. I am very glad that Dr. Schatz brought out, however, the giving of thyroid extract in adenoid conditions where we have a return of the adenoids. I have had only one case and it was a doctor's child, in which the adenoids had been removed three times. I was about to lose my reputation, but the child still kept closed up, he could not breathe and the adenoids kept returning about every four months. Finally I sent the child to Dr. Pfahler, thinking that perhaps there was an enlargement of the thymus. The report came back that the thymus was not enlarged and that possibly it was a thyroid enlargement. I administered the thyroid with a little strychnin and I am glad to say that in this case the result was that the adenoids did not appear again and the child improved mentally. This was six or eight months ago and so far there has been no return. There are two things that you have to consider and one is your thyroid gland and the other your thymus gland. Both of these will have a tendency to increase the likelihood of the return of adenoid tissue.

DR. SCHATZ (in closing): There should be a little caution in giving thyroid extract. During menstruation it is advisable to stop, in warm weather to give smaller doses than in cold weather, when it produces some weakness, to stop thyroid or give strychnin. One symptom of intolerance is pain in the back and nervousness. Watch for this and ask patient about these symptoms. In fact, I relied entirely on patient's story as to how she felt and on her weight. This patient cannot be reduced much in weight. I began to reduce the dose and found out what was the limit and got her a dose big enough to reduce symptoms and small enough not to reduce her weight.

Case Presentation of Gumma of the Tongue with Marked Blood Vessel Changes. Dr. Henry A. Laessle.

I regret very much that this patient I saw but twice. A man fifty-eight years of age came to me with pain on the right side of his neck and also complained of a swelling. When I looked at this growth I asked him for a history of venereal infection and I decided to have a Wassermann made, which came back 4 plus. After carefully examining the side of the neck, I found that he had a gumma the size of a walnut, about 1½", and also very hardened blood vessels, so I immediately gave him an injection of neosalvarsan. That seemed to produce a phlebitis. Five days after that, I noticed there was a thrombosis also, four or six inches in length. I told him to report to Dr. Eiman for a blood test, but I find that he did not report and he has not reported again to me. I found this to be an extremely interesting case for a man of fifty-eight years of age, but I regret that I cannot give any more detailed report. I would like to know why this phlebitis should happen and thrombosis happen after all the sterile precautions have been taken. Whether it was due to the thickening of the blood vessel wall or faulty technic or not, I do not know. I would like to know something about it. I have given salvarsan many times, but have never had this experience.

"Case Presentation Four Years After Operation for Large Cyst of the Frontal Lobe." Dr. Wm. G. Shemeley, Jr.

This case was presented before by Dr. MacKenzie in 1917. At that time Dr. MacKenzie presented the case to show the importance from the standpoint of aiding in making a diagnosis of intracranial complications and having a well made eye, ear, nose and throat examination. Since possibly some of you may not be familiar with the case, it might be well to run over some of the salient features presented in 1917. At that time the patient was brought into the office of Dr. MacKenzie. He had a very unsteady gait, requiring the support of a person on each side. He was very much emaciated, speech was slow. He complained at that time of severe pain in the right side of his nose, especially in the temporal and frontal regions. During his attempts at walking there was a tendency to pitch forward.

Presentation of Case, Four Years After Operation, for Large Subdural Cyst of the Frontal Lobe. Dr. Wm. G. Shemeley, Jr.

DISCUSSION.

DR. MILLER: It is rather interesting to see a case of this character after four or five years. I want to congratulate Dr. MacKenzie and his associate on the very careful and thorough examination of this patient and the excellent results that they obtained. This patient was supposed to have been thoroughly studied before he went to Dr. MacKenzie. I happened to be the first man who saw the case. This man was brought to me by the family physician because he had headaches and could not see. I watched him for about two weeks and found his choking symptoms increasing. He was then taken into the hospital and was examined by one of the most eminent ophthalmologists, who said that the case was one of albumen rhinitis. He had a Wassermann taken, blood counts made and X-rays made. We have numerous notes about this man. He was supposed to have been failing steadily. Neurologists could not find anything the matter with him. As far as doing anything for this man was concerned, we could not do anything for him. He then passed from my hands, to another nose and throat man possibly. I found nothing in his nose except some polyps, which I removed. He was then referred to Dr. MacKenzie and I found that he had operated on him. This man had evidently a traumatic condition. He gave a history of getting up at night to get something and tripped and fell all the way down stairs. His pictures, as I saw them first, were an absolute picture of an eye condition due to pressure, nothing else. The large veins, the narrow arteries and the swollen disc pointed toward a pressure. Of course, there was a possibility at the time of the early history of the case that some of these symp-

toms might have been brought on by sinus involvement, but still in the after study of the case, it proved that they had nothing to do with it. The man was lucky to have escaped from us and fall into the hands of Dr. MacKenzie.

DR. L. C. VATTIER: I happened to be present when this man came to Dr. MacKenzie's office. He had the typical findings of choked disc. I also had the pleasure of being present at the operation when the cyst was removed. I examined the man tonight and I should like to ask Dr. MacKenzie if the fields have been taken on this man. The discs look red and the veins a trifle smaller.

DR. GEORGE W. MACKENZIE: I have not examined the patient since the operation. I think there have been fields taken and the fields are considerably narrower.

DR. SHEMELEY (in closing): The question has been asked as to whether I had made a blind spot study of this man. I had not. There are some other phases of this case that we wanted to study a little longer. While this man is very willing to come down at all times to be studied, he lives in Kensington and it is a task for him to come down. The other question was that I did not mention about tension. He showed tension 25 in both eyes.

Throat and Ear Symptoms in Rheumatic Cases. Dr. Douglas Macfarlan.

DISCUSSION.

DR. HENRY A. LAESSLE: Dr. Macfarlan's paper has been an extremely interesting one. One thing I have noticed and that is that this type of case usually has a very severe sore throat during the night and is relieved during sunlight. Also in reference to the color of the mucous membrane and the pillars, it is a purplish red color. At the first offset of this condition, we find the mucous membrane quite red and dry. As far as the tinnitus is concerned, I firmly believe that is toxic. I noticed in cases we had in the hospital that if we gave cathartic in the course of a day or two, the tinnitus seemed to disappear and also a frightful pain that takes place in the throat. In conjunction, I dare say that most of these patients also give a history of spasm of one or the other of the upper eyelids. I have also used the alkaline treatment without the cathartic with negative results. I believe that if you examine the urine, you will find very much indican, a high specific gravity and total solids will also be increased.

DR. GEORGE W. MACKENZIE: I would like to emphasize what Dr. Macfarlan said in regard to lateral pharyngitis of rheumatic origin. The cases that I observed appeared as a tinge of blue redish ribbon behind the posterior pillars. The pain is worse at night and by conversation. During the day time it is better. These character of throats last for weeks and weeks.

DR. HARRY A. SCHATZ: I would like to take exception on one point. I have slides of the posterior lateral wall of the pharynx in which I can show Dr. Macfarlan that the lymphoid tissue is very prevalent in the lateral walls of the pharynx. Rheumatic pharyngitis is not a swelling of the lateral wall. It is simply a lymphoid condition, probably a hyperplasia.

DR. HENRY DINTENFASS: I would like to ask the question whether the external otitis in these rheumatic cases are like a furuncle or whether they are general like an ecyzema. It is surprising how quickly and easily these symptoms disappear without the use of local treatment, just by the alkaline administration.

DR. HENRY A. LAESSLE: My reason for bringing out that it was toxic is the fact that the tinnitus is significant with the pulsation and the indican and high specific gravity of the urine.

DR. DOUGLAS MACFARLAN (in closing): Dr. Laessle and MacKenzie said that the purplish tinge is typical. I have not noticed it. As to the hydro cathartic, these cases will be constipated and your laxative will relieve them, especially your alkaline. If the tinnitus is present (it does not have to be present) you will find your indican and high specific gravity in the urine. It strikes me that tinnitus is also toxic. The suggestion

that it might be the closing of the eustachian tube, it does not appear frequently enough. In regard to the adenoid tissue that Dr. Schatz mentions, of course, the whole naso-pharynx is an adenoid sheath. As these patients get older, the adenoid element is so negligible that when you find a patient forty years old with these irritations with none of the symptoms that you see in adenoid swelling, it seems impossible that the thing can be an adenoid swelling.

"Case of Acute Exudative Catarrh of the Middle Ear." Dr. W. S. Jones (by invitation).

In so much as I am a student at the University of Pennsylvania Graduate School of Medicine in the department of oto-laryngology, I am not presuming to bring anything new for your consideration, but simply try to call your attention to a disease of the middle ear which may be easily overlooked and which usually responds readily to treatment.

In my description I shall try, in so far as I am able, to follow the teaching of Dr. George MacKenzie, who has tried so diligently to teach each one of us to recognize a normal ear drum and know its anatomy.

Acute exudation catarrh of the middle ear may be called sero-mucous catarrhal otitis media or acute productive inflammation of the middle ear. In the middle ear there are two types of inflammation: (1) The destructive and (2) the productive type. We shall confine our attention to the productive type only, which is characterized by slight hyperemia and swelling of the mucous membrane of the middle ear and a discharge of sero-mucous exudate into the tympanic cavity. The process runs its course without perforation of the drum membrane, and either gets entirely well or leaves behind the foundation for that old friend of the otologist—chronic catarrhal otitis media.

Etiology. This condition is practically always secondary to pathological conditions in the naso-pharynx. The process extends from eustachian tube into the tympanic cavity. One of the principal predisposing causes is adenoid vegetation which lies so near the mouth of the eustachian tube. "Colds," rhinitis, sinusitis, tonsillitis, and pharyngitis are the most potent activating causes for blocking the mouth of the eustachian tube, thereby causing a tubo-tympanic catarrh to be set up.

Pathology. The blocking of the eustachian tube from an exudate or swelling of the mucous membrane causes an edema of the tympanic mucous membrane and an infiltration of round cells into the tissue. There is clear sero-mucous exudation thrown off into the cavity from the stimulation of the mucous glands.

Symptoms. The complaint which is most common is a sense of "fullness in the head." The patient will often place his little finger in the ear and shake the finger as though something was there and he was trying to dislodge it. Pain is rarely a noticeable symptom. "The more trifling the swelling and disturbance of hearing, the greater the sensation of fullness." (Politzer.)

The disturbance of hearing varies greatly,—from only slight impairment to a very noticeable reduction in hearing. The disturbance depends upon the character and amount of fluid. If the fluid in the middle ear is small in amount, the hearing will be only slightly impaired; if cavity contains considerable fluid the hearing is more appreciably impaired. If the fluid is thin the hearing may be increased by leaning the head forward. Gruber states that when the condition is unilateral the patient is often annoyed by the resonance of own voice in the diseased ear.

Subjective noises are quite variable. They may or may not be present. Crackling and popping in the ear may be noticed during the act of eating or swallowing.

Functional Test. There is a reduction in the ability to hear the low tones, while the high tones are heard normally. The Weber is referred to the diseased ear, provided the other ear is normal. The Schwabach is lengthened. The Rinne may be negative or positive. However, the results from the Rinne depend upon the amount of fluid in the tympanic cavity.

Otoscopic Picture. The normal drum is here shown for comparison with the picture as seen in acute exudative catarrh of the middle ear.

Rt. Drum. 1. Drum is intact. 2. Normal brilliancy and cone of light. 3. Grayish color. 4. Translucency enough to get slight image of the incus and posterior border of the handle of molleus. 5. Superior half—inferior half. Posterior half—anterior half. 6. Normal motility with Politzer bag and Siegel otoscope.

The appearance of the drum depends upon whether the tympanic cavity is filled or partly filled with exudate. First, in case the cavity is filled with fluid we see the drum has lost its luster and cone of light. It appears faintly yellowish gray. The yellow element is very slight. The incus is no longer seen and the hammer handle appears narrow. There is no bulging nor redness. On inflation we see bubbles appearing in anterior inferior quadrant and later the bubbles rise to top of tympanum.

When the tympanic cavity is only partly filled with fluid the picture is somewhat different. Here we see upper level of the fluid as a distinct grayish black line (nivean line). Below the line the drum has the peculiar grayish-yellow color and above the line it usually has its normal characteristics. There is no retraction, no bulging and no redness.

Treatment. Successful treatment consist in: (1) Make tube patulous. (2) Remove the fluid. (3) Treat the causative factor.

1. The eustachian tube is made patulous by the Politzer bag after applying cocain to mouth of tube. Sometimes it is necessary to use the catheter. If the fluid is not too thick it can be removed by Politzerization.

If improvement after Politzeration (and this is recognized by the appearance of bubbles and the lowering of the Nivean line in the middle ear), this treatment is continued until a cure is brought about. The treatment may be repeated daily or at longer intervals if it is found that the fluid doesn't return and there is a marked improvement.

If after two or three trials we are unable to remove the fluid a myringotomy is indicated. In such case a small piece of cotton is placed next to the drum and over the incision and then use the Politzer bag. The sero-mucous material takes hold to the fibers of the cotton, so to speak, and may be withdrawn with the cotton.

3. Any pathological condition in nose or pharynx should now receive treatment.

DR. MCNAMARA: I would like to say a few words. From the standpoint of having seen two cases in which we were able to demonstrate these changes occurring in the membrane, I would like to say something about them. In the first place, I would like to mention one little point that I think Dr. Jones happened to skip, and that is in cases in which we do not see the patient immediately after the beginning of the attack, that is, in which changes have gone on to the point in which the fluid within the tympanic cavity have become thick and mucoid, that when we do polleritize these cases, it is something to get these bubbles or to see them rise. In the second place, in the case in which you have a cavity which is completely filled with fluid, naturally you do not get the bubbles. We were very anxious to distinguish some of these cases after following them very closely. The first case that I saw was a case that at the time of history, she had been out playing golf, exposed to the weather, developed a severe earache, which lasted until about two and which was relieved. In the meantime, she had the usual family medicines. She complained of no further pain. From that time on, there was a feeling of stuffiness in the head which she described that one of her ears felt as though she had been in swimming. She also noticed that she was a little deaf in that ear. She came to my clinic five days after that and when I looked in I discovered this so-called nivean line, which was very distinct. It presented as a grayish black line. The membrane was not bulging, the color was not a normal luster and it had a so-called frostered glassy appearance. When we polliterized this case we were unable to see any diminution of the nivean line. Still we were sure that we got into that cavity. However, the case was seen two days later by one of the assistant professors, who polliterized the case again and he looked in and found the line had disappeared and the membrane was practically normal. We did not do any tuning fork tests on this patient.